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LECTURE I.

THE ERYTHRON IN HEALTH AND DISEASE.*

> "As regards the relative value of clinical and post-mortem observation in the diagnosis of cases of anæmia of doubtful nature, there is in my experience no doubt. For purposes of diagnosis I would rather see a case of severe anæmia during life than base my diagnosis on any blood changes or other features that might be presented in the form of reports."
>
> William Hunter, 1923.

The original duty of the Goulstonian lecturer was to dissect the body and to demonstrate his dissections. In the same spirit I shall try to draw a simple picture of that interesting tissue which is constituted by the red blood-cells and their precursors, and to give examples of its reactions in health and disease. Boycott has suggested the term *erythron* to comprise the circulating red blood-cells and the cells from which they arise. This term is valuable not only because of its brevity, but also because it emphasises the functional unity of the red cells and their precursors. The erythron may be aptly compared with an organ such as the skin. Just as in the skin we recognise the strata germinativum, mucosum, granulosum, lucidum, and corneum, so in the erythron we must think of the primitive cells evolving through megaloblast, normoblast, and reticulocyte to the mature erythrocyte. The interstitial tissue of the erythron is represented by the plasma, and by the fat and reticulum of the bone-marrow. Few people have a conception of the size of the erythron. The volume of the marrow cavity is estimated at 1400 c.cm., the total volume of the circulating blood at 4000 to 8000 c.cm. Even when no account is taken of the plasma, the size of the erythron can hardly be less than 2500 c.cm., so that it is an organ considerably larger than the liver. Escaping from the vessels at autopsy, or lying hidden in the recesses of the bones, it has too often escaped the attention of the morbid anatomist, and the pathology of the erythron remains to be written.

Boycott has shown that the erythron may, like other organs, undergo atrophy or hypertrophy, the one from increase of the oxygen tension, the other from its diminution, and he regards Osler's disease, polycythæmia rubra, as an obscure hyperplasia or perhaps a veritable tumour of the erythron. Following his lead we may speak of the congenital dystrophies of the erythron, the best known of which is acholuric familial jaundice, in which an abnormal and fragile corpuscle is transmitted from generation to generation. Less familiar to us but of the same hereditary type are the sickle-cell anæmia of negroes and the rare ovalocytosis. The anæmias which follow hæmorrhage or the lysis of the corpuscles by chemical or bacterial poisons are obvious examples of trauma to the erythron and are repaired according to the ordinary rules of pathology. Just as other organs are damaged by infections and toxæmias, undergoing parenchymatous degeneration or fatty change, so also the erythron with consequent anæmia. Familiar examples are the anæmia of typhoid fever or of chronic nephritis. Not only is the circulating blood impoverished, but the marrow is hypoplastic. In long-standing cachexias the hypoplasia may terminate in a peculiar gelatinous degeneration of the marrow, while a more acute poisoning, such as occasionally follows the injection of salvarsan, leads to complete aplasia of the bone-marrow. The erythron may be the seat of tumour metastasis, or be compressed and almost obliterated by the proliferation of the white cells in leukæmia.

Erythrogenesis and Erythrolysis.

So far we have applied to the erythron the same principles as we should apply to the consideration of the diseases of any other organ, and have discussed the banal effects of trauma, infection, and so on, effects which we are accustomed to describe as secondary or symptomatic anæmia. Most organs, however, have their special diseases, as examples of which I would cite Bright's disease of the kidneys, the cirrhoses of the liver, or the peptic ulcers of the stomach and duodenum. To understand these diseases an intimate knowledge of the structure and function of the organ at fault is necessary. It is not otherwise with the erythron, and if I seem to spend long in discussion of its structure and function, it is because I feel that such knowledge is essential for the understanding of its diseases. If we pause to wonder why the blood contains roughly 100 per cent. of hæmoglobin and 5,000,000 red cells per cubic millimetre, and how it is held constant at this level by the activity of the bone-marrow, we shall soon find ourselves engaged in speculations that seem to lead us far from the immediate problems of medicine. The hæmoglobin content of the blood is dependent on the oxygen tension of the inspired air, and on the need of maintaining a constant internal environment for the organism. The size and number of the red cells are such as to ensure the best utilisation of the hæmoglobin store of the organism, especially in the passage of the blood through the capillaries.[7]

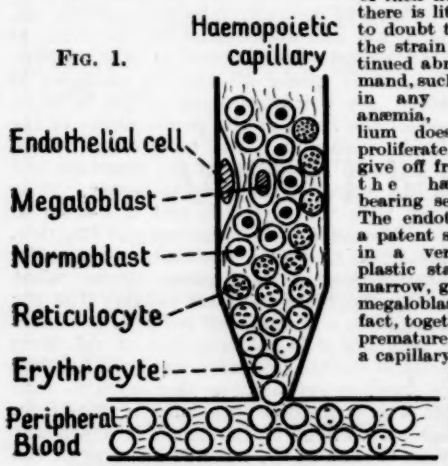
Red marrow is confined to the cancellous bones and the ends of the long bones. The vascular system of the bone-marrow is closed and the whole of the erythron, including the most primitive cells from which it arises, is intravascular. The capillaries of the marrow not only serve to maintain the circulation, but they also have a dual function in erythrolysis and erythrogenesis. The endothelial cells of the sinuses are phagocytic like those of the liver and spleen. Connecting the sinuses are the intersinusoidal capillaries in which the red cells are formed intravascularly, in contrast to the white cells which arise outside the vessels and migrate into the circulation when they mature. The endothelial cells lining the intersinusoidal capillaries give rise to the earliest megaloblasts by mitosis into the lumen. Successive divisions of the megaloblasts produce the erythroblasts, normoblasts, reticulocytes, and thus the red cells of the circulating blood. Developing within the capillary, the red blood-cells dilate the lumen and stretch the wall, but the capillary does not normally open to the circulation until the red cells are mature. This is because the membrane of the young cells is sticky and they adhere to one another. The stickiness persists till about the time when the reticular substance disappears from the cell. The plasma then filters

* The first Goulstonian Lecture for 1932 delivered before the Royal College of Physicians of London on March 1st.

between them, and they are swept into circulation. Just as the capillaries of the muscles and the kidneys vary greatly in activity with the activity of the tissue they supply, so also the capillaries of the marrow. In the marrow they offer not merely a reserve capillary bed, but the potentiality of an enormous hyperplastic response.

"In the normal, non-hyperplastic, unstimulated state, the erythroblastic level is the one from which the vast majority of the red cells come. Erythroblasts proliferate rapidly, and in the normal marrow are the youngest type of cell to be seen in all recognisable foci of red blood-cells. One megaloblast is conceivably sufficient to form the basis of an extensive focus of erythroblasts, which may be able to multiply quite indefinitely under normal conditions. Thus the endothelial cells not being called upon to augment the foci of developing red cells which can be maintained by the erythroblasts themselves, or at most only being stimulated to an occasional differentiation of megaloblasts from an isolated endothelial cell, commonly appear in the older, inactive phase. . . . Normal marrow has a considerable store of normoblasts which can then pass through the next phase of their maturation into erythrocytes by the extrusion of their nuclei. But there is little reason to doubt that under the strain of a continued abnormal demand, such as occurs in any secondary anæmia, endothelium does actively proliferate, and does give off free cells of the hæmoglobin-bearing series. . . . The endothelium of a patent sinus may, in a very hyperplastic state of the marrow, give rise to megaloblasts. This fact, together with a premature opening of a capillary, may explain the method by which immature cells get into the circulation."[5]

FIG. 1.



Schematic diagram of the erythron.

During the maturation process the nucleated erythroblasts elaborate hæmoglobin and they lose their nuclei before becoming adult red cells. Except for a small fraction of reticulocytes, which in health does not exceed 1 per cent., the circulating erythrocytes are all mature cells. The mature red blood corpuscle is as highly specialised and as "dead" as the stratum corneum of the skin. The reticulocytes are so called because vital stains render visible in them a network or reticulum, which is really a precipitate of remnants of the basophil cytoplasm of the immature cell. Reticulation is identical with polychromasia, though the vital stains display the basophilia better than eosin-methylene-blue; punctate basophilia is a modification of the same phenomenon. In the blood stream, or at body temperature in vitro, reticulocytes ripen into adult corpuscles, the process taking from a few hours to a few days, according to the amount of basophil substance they contain. When the delivery of red cells into the circulation is increased, as happens after a considerable hæmorrhage or in the regenerative phase of anæmia, the percentage of reticulocytes rises and may reach 50 per cent. or higher. Conversely, when the delivery of red cells into the circulation is diminished, as happens with artificial plethora in animals or in non-regenerative anæmia in man, the number of reticulocytes falls below the level of health. Enumeration of the reticulocytes is therefore a valuable method for determining the rate of blood formation,

When blood formation is excessively active, the more immature cells, normoblasts and megaloblasts, enter the circulation. Whenever the bone-marrow is hyperplastic nucleated red cells may appear in the peripheral blood; for it is then easy for them to be washed into the blood stream, especially when hæmopoiesis is not confined to the intersinusoidal capillaries and invades the sinuses. Hyperplasia of the marrow is by no means synonymous with increased blood formation, the reaction is often abortive and the cells fail to mature. This is a very important fact which I shall discuss in full later. I must now emphasise that nucleated red cells are a sign only of hyperplasia of the bone-marrow and are not evidence of increased blood formation unless they are accompanied by an increased number of reticulocytes. The coexistence of nucleated red cells and a reticulocytosis is of course unimpeachable witness of an accelerated delivery of red cells into the circulation, for the reticulocytes will ripen into adult red cells in the blood stream.

Having followed the red cells to their entrance as mature erythrocytes into the circulating blood, we must now inquire into their ultimate fate. Their life has an average duration of 30 days and it is not a whit less hard than that of the superficial cells of the skin. Anyone who has seen cinematograph films of the capillary circulation must have been impressed by the wear and tear to which the red cells are subjected. In Peyton Rous's dramatic words, they are finally threshed to bits. The terminal disintegration of the worn-out red cells can be watched under the microscope with the aid of a warm stage:

"A red cell puts out a long process and thereby becomes the so-called poikilocyte. At first the process does not move, then it begins to vibrate slowly, then faster and faster. This vibration marks the beginning of fragmentation. As the process vibrates, it gradually becomes thinner and thinner at some point and then separates. If the thinned-out place is at the base of a long process, the fragment becomes a rod, often looking like an irregular but yellow bacillus; if near the tip, it becomes a round or oval body. We have seen every variation in size and shape of these processes, up to an actual division of a red cell into two equal parts. . . . We interpret a poikilocyte as the first stage in the process of fragmentation, and the microcyte, in many instances, as a red cell reduced in size after some of its cytoplasm has broken off."[6]

Exercise, by increasing the wear and tear of the red cells, accelerates their disintegration. In anæmia the cells produced by the bone-marrow are less perfect and more easily worn out. For both reasons rest is an essential measure in the treatment of anæmia. I do not think lysis of the red cells occurs in health, their ultimate fate is always fragmentation. The fragments are taken up by the phagocytes of the reticulo-endothelial system and digested into simpler components. Iron and bilirubin are the most familiar of the disintegration products. It is well established that when large numbers of red cells are suddenly destroyed, the bilirubin in the blood and the excretions is increased and iron is deposited in the liver and other organs. About this there is not a shadow of doubt. On the other hand it is a serious error to argue in the reverse direction and to conclude that hyperbilirubinæmia and hæmosiderosis are always the result of increased blood destruction. Indeed the estimation of the rate of turnover and destruction of red cells is beset with difficulties.

The Dynamics of Blood Formation.

A fundamental question of hæmatology is the decision whether an anæmia is due to diminished

blood formation or to increased blood destruction. It is as important to decide this as to decide whether jaundice is obstructive or non-obstructive, or whether anuria is due to suppression or retention. Yet I do not exaggerate when I say that we have no reliable criteria for this purpose. It is desirable that the difficulties should be appreciated, for progress is much retarded when obscure anæmia is attributed without valid evidence to unproved hæmolysis or toxic depression of the functions of the marrow. Hæmatology will not be an exact science until we can measure the number of red cells, which enter or leave the circulation in the 24 hours, with the same accuracy as we can measure the protein metabolism or the work of the heart.

One of the breakdown products of hæmoglobin is the iron-free pigment bilirubin which, after circulating in the blood, is excreted in the bile and converted in the intestine into stercobilin, the form in which it leaves the body. The output of bilirubin can be accurately determined by estimating the stercobilin of the fæces; indirect evidence can be obtained from the bilirubin content of the blood and the urobilin of the urine, though the results are then affected by the functional condition of the liver and kidneys. Physiological experiment shows that there is a fundamental fallacy in trying to gauge the rate of blood destruction from the bilirubin output. When blood is destroyed, the bilirubin obtained from it is not quantitatively excreted. Bilirubin is not a waste product and much of it may be retained to be used over again in the manufacture of fresh red cells. The proportion retained is not a fixed quantity but depends on the needs of the organism.[25] From physiology we must conclude that the turnover of red cells might be increased without any change in bilirubin excretion.

The study of cases of anæmia brings to light even greater difficulties. In pernicious anæmia the excretion of bilirubin derivatives may vary from normal to 20 times the normal in different patients, who show the same profound degree of anæmia and are otherwise indistinguishable. Both Whipple and Jedlicka have calculated the level of the blood exchange in pernicious anæmia on the assumption that the bile-pigment excreted per diem was derived from the breakdown of red cells. Jedlicka calculated that the excretion of bilirubin derivatives may be 13 times as high in a patient with pernicious anæmia as in health, and that in extreme instances such a person must renew the whole of his blood in 21 hours. This is a reductio ad absurdum, for it is impossible for the blood to be renewed in so short a time.

In a subsequent paragraph I shall discuss recent work which proves that hæmolysis plays a secondary part in pernicious anæmia and that the primary cause of the anæmia is a retarded delivery of red cells into the circulation, in fact a much reduced turnover of red cells. It is profitable to compare pernicious anæmia with another metabolic disease, diabetes mellitus. Hyperglycæmia, deposits of glycogen in the heart and kidneys, and excretion of sugar in the urine do not mean that carbohydrate metabolism is accelerated but the very reverse; they are signs of inability to metabolise glucose in the absence of insulin. Similarly in pernicious anæmia hyperbilirubinæmia, deposits of iron in the viscera, and increased excretion of bilirubin derivatives are not because the blood breakdown is accelerated, but because the bone-marrow is unable to use these metabolites in the manufacture of fresh red cells, when the hæmatinic principle, which is elaborated in normal digestion and stored in the liver, is not supplied. Hyperbilirubinæmia and increased excretion of bilirubin derivatives thus come to be associated with a diminished blood exchange. I must leave further discussion of pernicious anæmia till later, but I think there is no doubt that on the one hand the turnover of red cells may be increased without any change in bilirubin excretion, on the other hand that the excretion of bilirubin derivatives gives no reliable information as to the rate of blood exchange.

Estimations of the number of reticulocytes have added much to our knowledge of the rate of blood formation in disease, but this test must not be used uncritically, and it needs collateral support. Reticulocytes appear in the blood stream when the marrow is irritated by a variety of substances, such as shellac and colchicin, and marrow irritation must be distinguished from a real increase of red cell production. I have seen reticulocyte counts of 6 per cent. in aplastic anæmia, though the absolute number was small, and this is not surprising when we remember that a few remnants of hyperplastic marrow are usually left in this disease. An increased delivery of red cells into the circulation may be unaccompanied by any rise in the number of reticulocytes, as when a transfusion is given at the beginning of liver treatment in pernicious anæmia.

I have stated these difficulties frankly because they have been so much ignored, but I cannot promise to solve all of them, for the simple reason that there are not enough facts. If we know little about the pigment metabolism in health and disease, we know still less about the other breakdown products of the red cell. The biochemist has devised exquisite chemical analyses of the plasma to ascertain the functions of the kidneys, the liver, and the pancreas, but until recently he has overlooked the chemistry of the erythron itself. The iron content of the viscera is still too often deduced from the microscopical appearances and the ferrocyanide reaction, though these methods were proved fallacious at the beginning of the century. The chemistry of the marrow is unexplored. Happily this has now become one of the active growing points in medical progress. We already known that the loss of the nucleus in the development of erythroblasts into erythrocytes increases the excretion of uric acid[23]; that the reticulocytes contain nuclear phosphorus which disappears when they mature[16]; that there are characteristic changes in the free iron[24] and the lipoids[18] of the serum in the different phases of anæmia. To such researches we look for help in the dynamics of blood formation. In the meantime the physician will do well to remember the wise words of William Hunter with which I have prefaced this lecture. Anæmia must be studied in the patient, and not merely in a droplet of his blood. The erythron does not work in isolation, but in close dependence on the other organs of the body and in subordination to the whole. A blood picture, or indeed a marrow picture, is only a symptom; it may appear as the result of very different causes, and it demands for its explanation the whole history and present state of the patient.

Hæmopoiesis and Anhæmopoiesis.

We see the importance of these points when we study the behaviour of the erythron in disease. Morphologically the changes are simple, consisting on the one hand of hypoplasia or aplasia of the bone-marrow, on the other of hyperplasia. Two degrees of hyperplasia may be described. Usually the number of erythroblasts is much increased, and the red marrow spreads into the shafts of the lo ng bones

a phenomenon which I shall call *the normoblastic reaction*. In more profound disorders not only are the bones filled throughout their length with hyperplastic marrow, but the level of differentiation is shifted back to an earlier stage and megaloblasts proliferate actively, even appearing in the peripheral blood, *the megaloblastic reaction*. Hypoplasia and aplasia of the marrow, which are obviously accompanied by diminished performance of function, need not detain us, but hyperplasia cannot be dismissed so swiftly. Functional analysis shows that hyperplasia may accompany both increase and decrease in the output of cells from the marrow. It is false to assume that hyperplasia necessarily means an increased turnover of red cells.

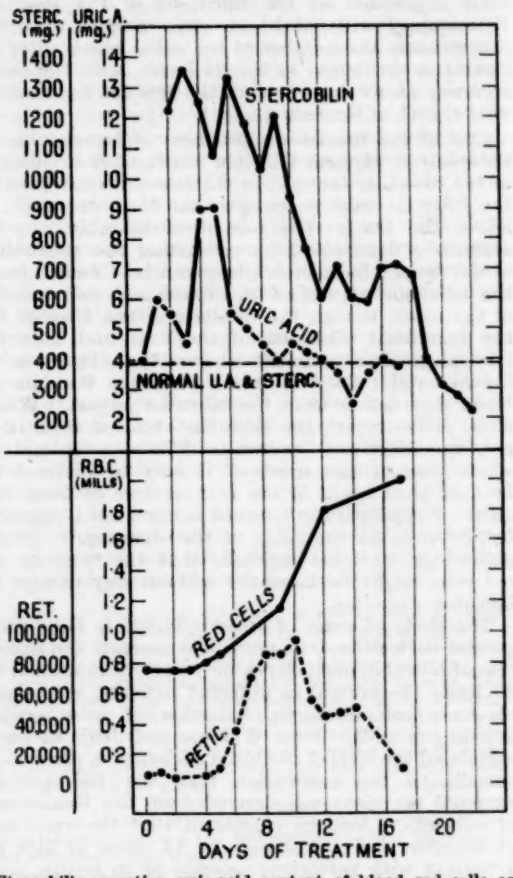
It is easy to see that the marrow must hypertrophy when cells are lost by hæmorrhage or hæmolysis, and according to the demands made, the ensuing reaction may be normoblastic or megaloblastic. None the less it is true that hyperplasia as often accompanies a diminished output of cells. After all, this is not a novel principle in pathology. Hyperplasia of the thyroid gland is present both in simple goitre, where the metabolism is normal or low, and in exophthalmic goitre, where the metabolism is high. Simple goitre is often due to iodine deficiency, a vain hyperplasia of the gland in the effort to overcome the deficiency. Similar changes occur in the erythron. When sufficient iron is not supplied, either from unsuitable diet or from diminished absorption from the alimentary canal, the erythroblasts multiply and the marrow encroaches on the shafts of the long bones, a normoblastic reaction, but the cells fail to mature and reticulocyte counts show that few cells are being delivered into the blood stream. Absence of the nitrogenous complex present in liver extract gives rise to a futile megaloblastic reaction, but again the delivery of mature red cells is diminished. It is as if a mass-production motor factory were unable to obtain an essential manufacturing part. Soon the assembly hall is crowded with unfinished cars, but few or none can be completed, and outside the street is empty of vehicles.

Let us summarise the evidence that pernicious anæmia is due to diminished production of red cells and not to increased destruction:—

1. In the early stages of the disease signs of hæmolysis are usually absent, although the morphological changes in the red cells are already clearly evident.
2. The observations of Ashby, and of Wearn, Warren, and Ames show that the red cell may survive as long in pernicious anæmia as in health.
3. The reticulocyte count is low, suggesting that few cells are leaving the bone-marrow, whereas in hæmolytic anæmias, such as acholuric jaundice, the reticulocyte count is high.
4. The excretion of bilirubin derivatives in the stools may be very different in different individuals with pernicious anæmia, although the hæmatological and clinical picture is the same.
5. The assumption that the bilirubin excreted is derived directly from hæmolysed red cells means an impossibly high level of blood destruction.
6. When remission occurs, the signs are those of increased activity of the bone-marrow and not of a diminution of pathological hæmolysis.

We have now a wealth of observations on the recovery phase of pernicious anæmia, and I have summarised some data given by Riddle and by Filo in Fig. 2. This is a composite diagram based on data from different cases and designed to show the time relation of the events which occur in treating a case of pernicious anæmia with liver. The first phenomenon noted is an increase in the amount of uric acid in the blood and urine. Control experiments on normal individuals show that this is not derived from the liver extract. It is due to the maturation of megaloblasts and normoblasts, with consequent loss of nuclear substance, which gives rise to an increased excretion of uric acid. The destruction of normoblastic nuclei appears to be the chief source of the endogenous uric acid in health, and during a remission in pernicious anæmia the amount of uric acid excreted in excess of the normal

FIG. 2.



Stercobilin excretion, uric acid content of blood, red cells, and reticulocytes in the remission of pernicious anæmia during liver treatment (from data by Filo and Riddle).

is proportional to the increase in number of the red blood-cells. In fact, an increase of a million red blood-cells per cubic millimetre is accompanied by the excretion of approximately 10 g. of endogenous uric acid in patients with pernicious anæmia during early remission. Next there is the well-known reticulocyte crisis. Its magnitude is directly proportional to the severity of the anæmia, and mathematical formulæ have been devised by which the "expected reticulocyte crisis" can be calculated from the initial red cell count with considerable accuracy.[23] The reticulocyte crisis is, in fact, directly proportional to the hyperplasia of the bone-marrow. Beginning with and following on the reticulocyte crisis, there is a steady rise to normal values in the number of mature red cells. All these facts are in harmony with the view that pernicious anæmia is due to a dysplasia or fruitless overgrowth of the bone-marrow which is repaired by liver extract, and I do not see how they can be adapted to the hæmolytic

theory of the disease. Finally, Filo has shown that the signs of increased activity of the bone-marrow, the reticulocytosis, and the rapid rise of red cells, may occur at a time when the stercobilin excretion is still considerably raised, and that it never happens that the increased stercobilin excretion disappears *before* the signs of regeneration of the red cells are seen.

I do not wish to deny that hæmolysis occurs in pernicious anæmia. There is in vitro evidence[3] of undue mechanical fragility of the megalocytes, and it is likely that these abnormal cells are not adapted for long survival in the circulating blood. The pathologist regards the phagocytosis of red cells and their debris as a hall-mark of pernicious anæmia, and it is rarely if ever absent in the severer stages of the disease. But this hæmolysis is secondary to the dysplasia of the bone-marrow, and it is inconceivable that there is so much hæmolysis as to account for all the bilirubin put out. The quality and distribution of the hæmosiderosis of pernicious anæmia is quite different from that of true hæmolytic anæmias such as acholuric jaundice. Some other explanation than hæmolysis must be found for the hyperbilirubinæmia and hæmosiderosis which are so constantly associated with the megaloblastic degeneration of the bone-marrow in pernicious and allied anæmias. As I have already stated, the disturbance of iron and pigment metabolism is probably the result of diminution of the anabolic activity of the bone-marrow. No such accumulation of iron or pigment occurs when erythrogenesis is arrested at the normoblastic level or in aplastic anæmia. It is in some way conditional on the megaloblastic level of dedifferentiation. I feel that the hæmatinic principles, which are necessary for the metabolism of megaloblast into normoblast, must also control the metabolism of iron and pigment, and that therefore the metabolism of iron and pigment is deranged contemporaneously with the megaloblastic degeneration of the bone-marrow, because both are the result of a common cause, and not because one is the effect of the other.

I have devoted considerable time to discussion of the mechanism of pernicious anæmia because it is the best-studied of all the anæmias, and because the principle of dysplasia of the bone-marrow is of fundamental importance in the classification of the anæmias. Tentatively we might divide the anæmias into two broad groups: first, those with hypoplastic or aplastic marrow, and, second, those with hyperplastic marrow. Then we must subdivide anæmia with hyperplastic marrow according as the hyperplasia is active or *hæmopoietic*, and passive or *anhæmopoietic*. The anhæmopoietic or maturational anæmias form in my opinion the most important of all the groups of diseases of the erythron, and I propose to devote the whole of my second lecture to them. Before concluding this lecture on the physiology and pathology of the erythron, I should like to say a few words first about splenomegaly in anæmia, and then about the size of the red cells in anæmia.

Splenomegaly.

With the exception of aplastic anæmia, all the chronic diseases of the blood-forming organs tend to produce enlargement of the spleen, whether they involve initially the red cells, white cells, or platelets. On this account the coexistence of anæmia, leucopenia, and splenomegaly never justifies an immediate diagnosis of splenic anæmia, but for lack of further investigation and observation I have several times seen aleukæmic leukæmia, essential thrombopenia, and simple achlorhydric and allied anæmias masquerade under this too comprehensive diagnosis. Splenic anæmia is in reality a rare disease. Enlargement of the spleen and anæmia are more commonly the result of an independent disease of the blood-forming organs than of splenic anæmia. It is now becoming accepted that splenic anæmia is always the result of high pressure in the portal circulation,[18] and that in the great majority of cases this is due to primary disease of the liver, though the morbid change in the liver may be visible under the microscope only.[15] The anæmia is the result of the raised portal pressure, as has often been shown in experimental animals.[21] Identical anæmia occurs in Laennec's cirrhosis of the liver.[17] It is unwise to diagnose splenic anæmia in the absence of signs of raised portal pressure or of liver damage without the most careful circumspection.

The Size of the Red Cells.

If splenic anæmia has often been diagnosed too freely, the reverse mistake has been made about pernicious anæmia, which some hæmatologists have tried to preserve in almost sacred isolation. Pernicious anæmia is not a unique disease, and similar changes occur in the blood in a variety of conditions. The anæmias of sprue and of *Bothriocephalus latus* infestation are closely akin to pernicious anæmia. Our President has described instances of megaloblastic alteration of the bone-marrow in the crises of acholuric jaundice.[4] In both hæmopoietic and anhæmopoietic anæmia the hyperplasia of the marrow may be normoblastic or megaloblastic, and of this fact I shall give many examples in my subsequent two lectures. The appearance of megaloblasts in the marrow and of megalocytes in the peripheral blood is not a specific disease, but a reaction which may occur in a host of different conditions. A disease is "the whole consequence of a conflict between man or animal and the noxious agencies in his environment,"[26] and in the definition of disease entities the clinical and biological features must always be more important than the blood picture.

The reactions of the erythron, which I have described as aplastic, normoblastic, and megaloblastic, can be roughly correlated with variations in the size of the cells of the peripheral blood. The size of the red cells is estimated either by the Price-Jones curve, which is based on the diameter of several hundred red cells, or by the hæmatocrit. The Price-Jones curve has the great advantage of showing the degree of anisocytosis, which is not revealed by the hæmatocrit. The hæmatocrit measures the volume of the cells and may detect changes which are not evident from measurement of the diameter alone. The red cells maintain a very constant size in health, their coefficient of variation being less than one-third of that of the red cells or hæmoglobin. The size of a man's red cells, measured under standard conditions, is as characteristic and permanent as his finger-prints or his electrocardiogram. In disease the mean diameter of the red cells may change and the degree of anisocytosis increase. Price-Jones has recently constructed a curve which embraces all the possible variations in size of healthy red corpuscles. Cells falling outside this ideal curve are pathological. In pernicious anæmia the mean diameter of the red cells is increased and many fall to the right of the ideal curve. They are pathological megalocytes, indicative of abnormal erythropoiesis. On complete and successful treatment with liver, the curve shifts back to the normal position and the pathological

megalocytes disappear. In chronic hæmorrhage, and in simple achlorhydric and allied anæmias, the mean diameter of the red cells is diminished and many of the cells fall to the left of the ideal curve. They are pathological microcytes, and their appearance likewise is suggestive of abnormal erythropoiesis. Here the curve returns to the normal position and microcytes disappear on successful treatment with iron.

Curves identical with those of pernicious anæmia have been obtained from cases of *B. latus* infection,[11] sprue,[11] chylous diarrhœa,[12] and the hæmolytic anæmia of pregnancy,[10] and it would appear that they can be obtained whenever there is megaloblastic degeneration of the marrow. As regards the microcytic anæmias, it is noteworthy that there is microcytosis both in chronic hæmorrhagic anæmia and in simple achlorhydric anæmia, two conditions in which there is normoblastic hyperplasia of the marrow. The whole story of these variations in size of the red cells is not yet told and the time is hardly ripe for generalising. Price-Jones and his associates have shown that the curves obtained in certain non-Addisonian hæmolytic anæmias resemble that of Addison's anæmia in the increase of the mean diameter of the cells but differ from it in the symmetry of the curve and the absence of anisocytosis.[9] Again, when we consider the appearance of pathological microcytes in the blood, we must remember that some of them may be fragmented normocytes and not preformed microcytes. Finally, the reactions of the erythron are not always pure, and both megaloblastic and normoblastic reaction may occur simultaneously, so that pathological megalocytes and microcytes appear in the same blood. Even more often, a megaloblastic or a normoblastic reaction is accompanied by more or less aplasia of the marrow. With these cautions in mind, we may make the broad generalisations that megaloblastic hyperplasia of the marrow, whatever its cause, is associated with a *megalocytic or macrocytic anæmia*. Normoblastic hyperplasia of the marrow, whatever its cause, is associated with a *microcytic anæmia*. Aplasia and hypoplasia of the marrow, inasmuch as they produce a general depression of the erythron rather than a disturbance of the mechanism of erythropoiesis, cause little change in the size of the red cells and are associated with a *normocytic anæmia*.

This method of classifying the anæmias has been very fully developed by Wintrobe, Haden, and others with the hæmatocrit technique. Wintrobe's cases fell into the following four groups[29]:—

1. The *macrocytic* anæmias, which include cases of pernicious anæmia, sprue, and the pernicious anæmia of pregnancy, and which are characterised by an increase in the mean size and hæmoglobin content of the red corpuscles.

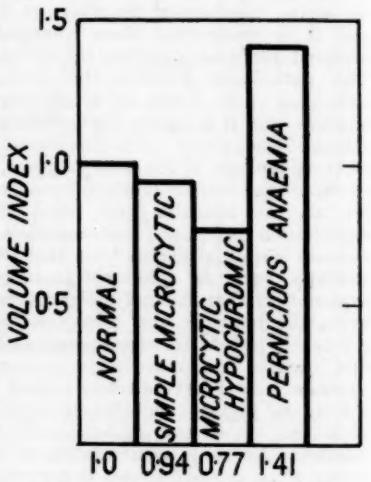
2. The *normocytic* anæmias, which include cases of acute blood loss, malarial infection, and anæmia of the aplastic or semi-aplastic type, and which are characterised by a reduction in the red cell count without any change or at most only a slight increase in the size and hæmoglobin content of the red corpuscles.

3. The *simple microcytic* group includes the most commonly observed forms of anæmia, the anæmia associated with chronic infections such as bronchiectasis and typhoid fever, chronic intoxications such as cardiorenal disease, and carcinomata in which there is no bleeding. There is a moderate reduction in the corpuscular volume and hæmoglobin, but at no time is this decrease as great as the reduction in the number of red cells.

4. The *microcytic, hypochromic* group, which includes the anæmia of chronic hæmorrhage, hookworm infestation, and simple achlorhydric and allied anæmias, is distinguished by a marked reduction in the volume of the red corpuscles and a decrease in their hæmoglobin content, which is even more marked than the decrease in size.

Very similar results were obtained by Haden, whose data I have used for Fig. 3. More work must be done before we can properly correlate the size of the red cells with the changes in the marrow. From what we already know of the marrow in infections and toxæmias, it seems likely that in group 3 there is a varying mixture of normoblastic hyperplasia and aplasia, which explains its position between the normocytic anæmia of aplasia and the frank microcytic anæmia of normoblastic hyperplasia. It may be noted in passing that the red cells never become more saturated with hæmoglobin than they are in health, and that a high colour-index is always a sign of megalocytosis. On the other hand, it is quite possible for the corpuscular concentration of hæmoglobin to fall, and a low colour-index may be due to low hæmoglobin concentration or hypochromia, as well as to microcytosis.

FIG. 3.



Volume index of red cells in health and in anæmia.

A given lesion may always produce the same change in the erythron—for instance, hæmorrhage always leads to a normoblastic reaction—but we shall not be surprised to find that the same agency, working under different conditions or with different intensity, may at various times produce any of these reactions. For example, sprue may begin with a normoblastic reaction associated with a microcytic anæmia, progress to a macrocytic anæmia with megaloblastic marrow, and terminate in aplastic anæmia. Nor shall we expect the reaction always to be pure in type, any more than tumours are sharply divisible into innocent and malignant. The anæmia of gastric achlorhydria sometimes has features of both normoblastic and megaloblastic response and the tropical anæmias show a mixture of megaloblastic reaction and aplasia. All this must persuade that a blood picture is not a disease, and in discussing anæmia in the two subsequent lectures, I shall work as far as possible on a clinical and ætiological basis.

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ELECTROCARDIOGRAPHIC CHANGES DURING TREATMENT OF SEVERE DIABETES.

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ASSISTANT PHYSICIAN, CHARING CROSS HOSPITAL; PHYSICIAN, ROYAL WESTMINSTER OPHTHALMIC HOSPITAL.

The present investigations concern electrocardiographic changes during the treatment of severe diabetes. The experimental method is outlined, and the results are tabulated and described. Serial electrocardiograms from typical and special cases are reproduced. The metabolism of the diabetic heart is contrasted with that of the normal heart and finally the possible mechanisms underlying the electrocardiographic changes occurring in diabetes are considered.

It was thought that the metabolic and arterial disorders occurring in diabetics might either independently or in conjunction be reflected in the electrocardiogram. In the course of treatment by diet or diet and insulin the defects of metabolism are largely abolished. Nor are ischæmic processes consequent upon, for example, early atheroma necessarily ineradicable, for Aschoff has demonstrated that this intimal degeneration is, up to the stage of fatty deposition, a reversible process. It was therefore considered most profitable to study electrically the heart in diabetics undergoing treatment with the aim of revealing modifications in the electrocardiogram.

One or two studies upon lines contiguous to these have already been made. Blitzsten and Schram made an electrocardiographic examination of 70 diabetics of all grades. In only six patients was an attempt made to trace the electrical changes occurring during treatment. Notching of the Q R S complex was found in 30 per cent. of the group, and all the investigations pointed to an increased voltage in severe hyperglycæmia, the amplitude of the deflections falling as the blood-sugar diminished. No mention of the T-waves is to be found in their paper. An electrocardiographic study of 123 diabetics was made by Hepburn and Graham who found that in 56 there were significant electrocardiographic changes, but in only 23 of these patients was more than one record obtained. No attempt was made by these authors to trace successive changes during stabilisation by dietetic or insulin treatment, nor were tracings published by them. Other electrocardiographic investigations such as those of Weichmann and of Middleton and Oatway have been concerned chiefly with hypoglycæmia, while Stepp and Parade have studied the influence of insulin rather than the effect of diabetes itself. Further reference will subsequently be made to these studies.

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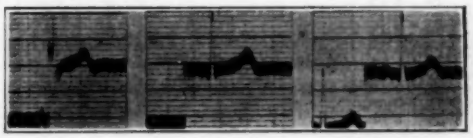
EXPERIMENTAL METHOD.

The patients selected for observation were those suffering from a severe grade of diabetes. In all the patients at the time of the first electrocardiogram there was considerable hyperglycæmia with glycosuria and variable degrees of ketosis, but actual or threatened coma was never present. In no case was there evidence from the history or the physical examination of cardiac or other additional disease, with the exception of variable degrees of arterial thickening and hypertension which were common among the older patients. Nor were patients showing glycosuria of doubtful origin, such as renal glycosuria, included. The initial tracing was usually taken just after admission to hospital and always before any dietary or other treatment had started. Subsequent records were obtained at intervals ranging from two to seven days, and usually after discharge from hospital further electrocardiograms were taken at intervals of two or three weeks, and later, months.

Fig. 1 (Case 6).



1 week. 5 weeks.

Successive records (lead I.) showing standardisation and increased amplitude of T-wave after one week of treatment.

The experimental group consisted of 20 patients whose ages ranged fairly evenly from 10½ years to 63 years. The duration of the illness in some of them could not be accurately ascertained as investigations were begun at the time of the first diagnosis. In others, presenting themselves with severe uncontrolled diabetes, the disease had started years previously but had never been adequately treated.

Full clinical and biochemical data were obtained in each case. For each patient the state of the urine was recorded both at the time of every electrocardiogram and during the intervals. Every care was taken to procure accurate and comparable electrocardiograms, with the minimal resistance in the patient. The usual standardisation of 1 cm. deflection for one millivolt was actually recorded for each lead in the majority of electrocardiograms (Fig. 1). These serial records were subsequently studied in relation to the changing clinical condition of the patient.

RESULTS.

The patients investigated are listed in the Table in order of age, starting with the youngest. They are arbitrarily separated into two age-groups, those below the age of 40, and those above. It is seen that in the earlier age-group the duration of the disease had been generally one year or less, while in the older patients investigated the disease had been

present usually for two years or more. The alterations in the component parts of the electrocardiogram are shown in each instance. The first three columns are self-explanatory. Absence of change is throughout indicated by a dash. The notation employed in regard to the T-waves is as follows: a slight increase in amplitude in successive electrocardiograms is indicated by (+), while pronounced increases are shown as (++). The sign (+) is also employed to denote the abolition of a previously inverted or diphasic T-wave, while (++) indicates the appearance of an upright T-wave in the place of one previously inverted or diphasic.

Alterations in the P-wave.—Where any change during treatment was observed it was a diminution. This was found in 6 of the 20 patients

Alteration in P-R interval.—No alteration in the P-R interval was discovered in any case. In some

Table showing the various changes occurring during treatment in the electrocardiograms of a series of 20 diabetic patients.

Case	Age.	Duration.	Alteration in— (*a*) P-waves.* (*b*) Q R S complexes.	Progressive changes taking place in— T_1	T_2	T_3
1	10½	6/52	(*a*) Diminishing.	+	—	—
2	18	1/12	—	—	—	+
3	19	2/12	—	+	++	++
4	22	6/12	(*a*) Diminishing.	—	+	++
5	23	2/12	,,	+	+	+
6	28	7/12	—	+	+	+
7	32	1 yr.	(*b*) Slurring diminishing.	+	++	+
8	33	3/12	,,	—	—	+
9	35	10/12	(*a*) Diminishing.	—	+	+
10	38	6 yrs.	—	+	+	—
11	39	1 yr.	—	++	++	++
12	46	8/12	(*b*) Slurring diminishing.	—	—	+
13	47	2 yrs.	(*b*) Slurring unchanged.	—	—	—
14	55	1½ yrs.	—	+	+	+
15	55	5 yrs.	(*a*) Diminishing.	—	—	—
16	58	6/12	(*a*) Diminishing. (*b*) Slurring unchanged.	+	+	—
17	59	2 yrs.	—	—	—	—
18	60	12 yrs.	—	—	—	—
19	61	4 yrs.	—	—	—	—
20	63	2 yrs.	—	—	—	—

* Alteration in P–R intervals: Nil in each case.

instances it was difficult to compare accurately the P-R interval on account of the somatic tremor present in the earlier electrocardiograms.

Alteration of Q R S complexes.—Slurring or splintering of the Q R S complexes was observed in five patients, both young and old (Cases 7, 8, 12, 13, and 16). In the first three of these (the younger patients) this deformity of the Q R S complexes either diminished or disappeared.

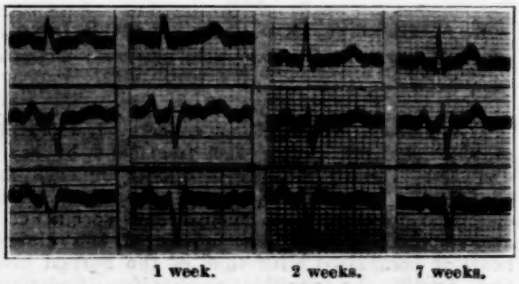
Alterations in the T-wave.—Taking these end-deflections as a whole it was noticeable that modifications occurred more readily in lead III. than in the other leads, and more readily in lead II. than in lead I. Further, it was found that T-wave alterations, whichever lead was considered, were far commoner in the younger age-group than among the older patients, while if any variations in the amplitude of the T-wave were found in the latter group, they were always slight.

The alteration most commonly observed in the T-wave during treatment was an increasing amplitude. This occurred mostly in leads I. and II. (Fig. 2). Another frequent modification was the substitution of an upright T-wave for one previously diphasic or inverted, this change being seen very often in lead III. and occasionally in leads I. and II. (Fig. 3, lead III.).

Changes in T in lead I.—Of the series of 20 patients only two showed initial inversions of T_1 which later became upright (Figs. 3 and 4). In these cases the restoration of a normal T-wave was gradual, occupying about seven weeks in Case 3, and over three months in Case 11. Increasing amplitude of T_1 during treatment was found in seven other patients.

Changes in T in lead II.—In lead II. also, only two patients showed inversion of the T-wave;

FIG. 2 (Case 7).



1 week. 2 weeks. 7 weeks.

Female diabetic, aged 32. First ECG taken before treatment. Successive records during treatment show, in leads I. and II., increasing amplitude of T-waves, which are finally considerably increased.

these were the patients already mentioned in whom T_1 was also inverted. Treatment restored T_2 to a normal sense and shape more readily than in the case of T_1, the change occupying a week or two (Figs. 3 and 4). Increasing amplitude of T_2 during treatment was found in eight other patients.

Changes in T in lead III.—In 9 of the 11 patients under the age of 40, and in 11 of the whole series, changes took place in T_3 during treatment. This change in seven instances was the substitution of an upright T-wave for one previously diphasic (Fig. 4). In the remaining four patients, increasing amplitude of an upright T-wave or the conversion of a frankly inverted T-wave to the upright form were noted. It was observed that these alterations in T_3 occurred readily, often within a few days of the institution of treatment. The form of T_3 was always apt to vary for no obvious reason (Fig. 4).

Effect of insulin.—In several patients the changes in the T-waves described above developed progressively, and later, after a few weeks, some diminution of the amplitude of the waves would occur (Figs. 1 and 5). The secondary changes seemed possibly to bear a relation to the use of insulin which is known to cause a reduction in the amplitude of the T-wave. Further reference to this point will be made below.

COMMENTARY.

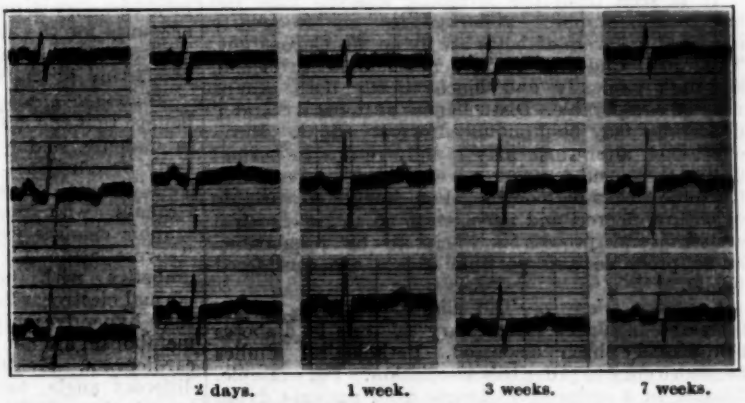
Among the complications to which diabetics are subject, arterial disease is one of the most formidable. The association of these diseases has been recognised for the past 50 years, but it is not generally appreciated how intimately they are inter-related. Joslin (1928) estimates that arterio-sclerosis in its various forms is responsible for half the deaths in diabetics to-day, while Allen claims that it is demonstrable in every diabetic past middle life in whom glycosuria has been present for ten years or more. Studies of patients over 40 years of age by Bowen and Keonig showed that nearly two-thirds were afflicted with arterial disease. A corresponding proportion of a group of 53 diabetics observed by Heitz gave evidence of early arterial degeneration on oscillometric examination.

But it is not only in the older diabetics that arterial disease supervenes. Shepardson in a radiological investigation of 50 patients of at least five years' standing, and whose average age was 23·4 years, found arterio-sclerosis in the large vessels of the lower extremities in 18. Similar results were obtained by White and Hunt who demonstrated by X ray degenerative changes in the arteries of the legs in 9 out of 48 children examined. The average age of the affected children was 15·8 years, and in them the disease had been present for an average of 6·3 years.

There is no lack of evidence that the coronary arteries are often involved in degenerative processes in the course of diabetes. Indeed, the frequency with which angina pectoris and coronary thrombosis occur in diabetes is a commonplace observation. Levine (1922) found that 7 per cent. of patients with angina pectoris had previously well-defined diabetes, while in his study (1929) of 145 patients suffering from coronary thrombosis, he found glycosuria or a conclusive history of diabetes in no fewer than 23·7 per cent. In these circumstances, and recalling the early age at which advanced arterial changes have been recorded, myocardial damage might well be expected even in young diabetics and in those free from cardiac pain. Joslin (1930), writing of arterio-sclerosis in the youthful diabetic, quotes the case of a girl in her early twenties who, after a week's premonitory symptoms complained suddenly of "pain in the heart" and died within five minutes. He further refers to two male diabetics aged 30 and 33 who died of coronary thrombosis and angina, the diagnosis in both cases being confirmed by autopsy.

The normal heart muscle stores glycogen, and according to Starling this substance is specially abundant in the conducting tissues. It seems possible that this fact may be connected with the immunity of the diabetics studied in this series from defects of conduction. That the profound disturbances of metabolism occurring in diabetes seriously involve the heart muscle was shown by Cruickshank and Patterson who demonstrated that the diabetic heart only utilises half as much glycogen per gramme per hour as the normal. It might therefore be expected that gross perversions of the normal chemical and electrical cycles taking place with each heart beat might occur even in diabetics having little or no general or coronary arterial disease. That grave disturbances of myocardial function do occasionally result from interference with carbohydrate metabolism has been shown by the studies of Hetényi, of Modern, and of Turner, who have recorded the occurrence of anginal pain as a result of hypoglycæmia in the course of treatment by insulin. Under the term "insulin angina" Parsonnet and Hyman have described the onset of cardiac infarction as an immediate sequel to insulin therapy in seven patients. The fact that in four of these the blood-sugar did not fall below the usually accepted margin of safety shows that hypoglycæmic shock was not the cause of

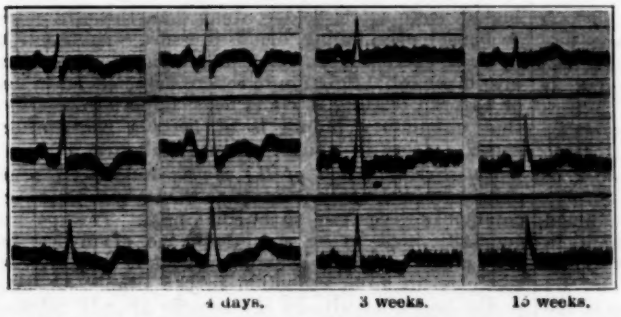
FIG. 3 (Case 3).



2 days. 1 week. 3 weeks. 7 weeks.

Female diabetic, aged 19. First ECG taken before treatment; note inverted T-waves. Successive records show gradual restoration of T-waves to normal form and direction in all leads after seven weeks of treatment.

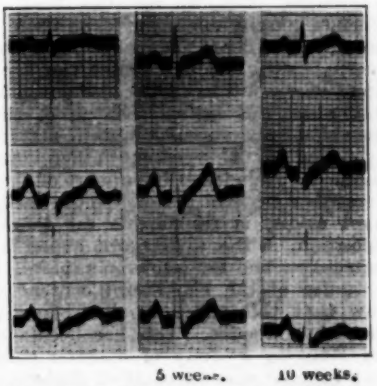
FIG. 4 (Case 11).



4 days. 3 weeks. 15 weeks.

Female diabetic, aged 39. First ECG taken before treatment shows concise inversion of T-waves, their form in leads I. and II. suggesting "coronary" T-waves. Later records during treatment show gradual reversion to normal after 15 weeks.

FIG. 5 (Case 14).



5 weeks. 10 weeks.

Male diabetic, aged 55. First ECG taken before treatment. After five weeks treatment the amplitude of the T-waves is increased in all leads. A lessened amplitude after ten weeks may be related to the action of insulin.

death. Apart from the production of anginal symptoms, there seems no doubt from the experience of these and other workers that heart failure also may be precipitated and accelerated in diabetics with myocardial disease by the use of insulin.

THE CAUSE OF ELECTROCARDIOGRAPHIC CHANGES.

It is seen from the foregoing analysis that the electrocardiographic abnormalities so frequently observed in severe diabetes are by no means constant. They are found in some patients and not in others, although clinically and biochemically both groups may be comparable. Disturbances of the Q R S and T deflections may be pronounced or very slight. Moreover, such alterations may rapidly return to normal during treatment or progressive changes may continue for some weeks or months. For changes so variable in nature, degree, and persistence, no single chemical or structural process is likely to be responsible. Some probable and possible causes are considered below.

Coronary sclerosis.—The clinical evidence for an increased incidence of coronary sclerosis in diabetes has already been discussed. Pathological studies have also gone to prove this association. For example, Blotner in a series of 77 hearts from patients dying of diabetes, found advanced disease of the coronary arteries in 45 per cent., as contrasted with a 21 per cent. incidence among 450 non-diabetic hearts. He also observed that cardiac infarction had been the cause of death in 10 per cent. of these fatal cases of diabetes. Obviously, in many diabetics of long-standing, and especially in those past middle life, there is established thickening of the coronary arteries often with irreversible intimal degeneration, and there can be no doubt that such changes with the consequent ischæmic myocardial fibrosis may account for gross and persisting electrocardiographic abnormalities.

Coronary intimal fatty deposition.—That transient intimal deposition of fat may occur in children and young adults is well known. In a discussion of this occurrence, Aschoff remarks that the "process depends purely upon the deposition of cholesterol esters." He quotes Mönckeberg, who in autopsies on 79 healthy soldiers under the age of 30 years, found aortic intimal fatty change in 57. It is held that while this intimal change of the "ascending period" of the vascular system may retrogress, the involution of a senescent atheromatous patch is absolutely excluded. In support of the view that an excessive concentration of lipoids, particularly cholesterol esters, is responsible for this intimal fatty deposition, is the well-known experimental work of Anitschkow and Chalatow on rabbits.

Studies of the cholesterol content of the blood show that this substance is frequently present in excess in diabetics. This has been shown by White and Hunt in a study of diabetic children, and by Gray in a large series of adult diabetics. Nor is the excess of cholesterol confined to the blood alone, for it has been shown to be present in unusual amount in the aorta and peripheral arteries in diabetics. Labbé, Nepveux, and Heitz found the increase to be 60 per cent. in the aorta and approximately six-fold in the tibial arteries. Since the lipoid concentration of the blood tends to be greater in severe diabetes the liability in the latter condition to fatty intimal deposition should be greater. The influence of hypercholesterolæmia in producing arterial damage remains questionable in view of the fact that the pronounced cholesterolæmias of jaundice and nephrosis are unaccompanied by arterial change. However, such cases are not strictly comparable with high-grade diabetics who have in addition a severe ketosis.

While electrocardiographic abnormalities that change within a day or two of the disappearance of hyperglycæmia and acetonuria might scarcely be ascribed to the retrogression of a fatty intimal change in a coronary artery, it is possible that this process accounts for the slow disappearance of gross abnormalities (Figs. 3 and 4) and for the gradual abolition of slurring in the Q R S complexes.

Abnormal metabolic products.—It is tempting to believe that the flattening or inversion of the T-waves in diabetes is due to the circulation of some toxic body or abnormal metabolic product in the blood. This possibility was discussed by Hepburn and Graham, who remarked that such electrocardiographic changes were not found in the few cases of "severe acidosis" studied by them, while many diabetics with considerable hyperglycæmia had normal electrocardiograms. They further concluded that hyperglycæmia and the products of perverted metabolism are not the cause of the abnormal electrocardiogram. Evidence on this matter has, from a different angle, been brought by Taterka, who has traced the electrocardiographic changes occurring in diabetic coma. His records were taken immediately after the institution of insulin and other treatment. He found a general lowering of all excursions, particularly of the T-wave, which was frequently diphasic or completely negative. These changes disappeared after recovery, even though high insulin administration was being continued. This fact, and the usual latency in the appearance of the electrocardiographic changes produced by insulin therapy apart from coma led Taterka to believe that the abnormal electrocardiograms could not have been due to the administration of insulin, but that a toxic myocardial damage due to acidosis must have been the cause. He states that comparable changes may be found in some infective diseases.

It has been shown by Dodds and Robertson that the theories of ketosis and acidosis are inadequate to explain the occurrence of coma in diabetes, since many non-comatose patients were found to have a higher aceto-acetic acid content than those in coma. A persistently falling blood pressure was found to precede death in coma. The present investigations into myocardial function in severe diabetes and those of Taterka relating to coma lend support to the view that circulatory failure is at least a contributory cause of death in many comatose diabetics.

Defective nutrition of the myocardium.—An acute deficiency in the blood-supply to the myocardium very commonly gives rise to anginal pain. This pain is generally held to be a consequence of myocardial anoxæmia, but there is evidence that it may alternatively result from reduction in the supply of sugar to the heart, as witness the records already referred to above (Hetényi; Modern; Parsonnet and Hyman; Turner) of angina pectoris precipitated by the use of insulin whether in normal or excessive amounts. Since deprivation of oxygen or of sugar may precipitate identical symptoms, there seems, by analogy, no reason to believe that they may not cause similar electrocardiographic changes. Acute anoxæmia has been shown by Ward and Wright to cause reduction, abolition, or inversion of the T-wave in lead II., while comparable changes have been recorded by Greene and Gilbert in chronic anoxæmia. It seems, therefore, not unreasonable to infer that the changes in the T-wave may be due to the reduction in the

sugar-utilisation of the heart, that has been shown to occur in diabetes (Cruickshank and Patterson).

Such a mechanism might account for changes which are only completely rectified after some weeks of stabilisation (Figs. 3, 4, 5), as well as for those which disappear more rapidly (Fig. 2). The form of the T-wave deviations in Cases 3 (Fig. 3) and 11 (Fig. 4) are of significance in this connexion. In both of these patients the T-wave was strikingly affected, and in each case there was either flattening or inversion in all leads. Such an effect does not suggest a coronary origin, for, as Parkinson and Bedford have shown, where depression of the R-T interval or T-wave in lead I. is the sequel to coronary obstruction, the change, if any, in lead III. must be in the direction of elevation. The T-wave deformities in the diabetic suggest rather an intimate parenchymatous change. The rectification of such changes of long standing might well take some weeks of dietetic and insulin treatment. A parallel may be observed in the myxœdematous patient who commonly shows inversion of the T-wave in all leads. These electrocardiographic anomalies, like those of the diabetic, do not suggest a coronary basis, nor is arterio-sclerosis the rule in myxœdema. Treatment by thyroid medication will, after some weeks or months, reproduce a normal electrocardiogram in myxœdema, just as appropriate treatment in the diabetic soon restores the normal nutrition and activity of the myocardium, thereby restoring the normal electrocardiogram.

It is profitable also in this connexion to compare the metabolic and electrocardiographic changes observed in hyperglycæmia with those of hypoglycæmia. These two conditions are the extremes of the blood-sugar scale; in most metabolic spheres they offer a sharp contrast, and yet the effect of both is a deficiency in the utilisation of carbohydrate. When there is sudden hypoglycæmia after previous prolonged treatment with insulin the tissues are not supplied with sufficient sugar for their needs, while in hyperglycæmia a glut of sugar is generally accompanied by an inability of the tissues to utilise it. Deficient utilisation of sugar is thus a feature of both conditions. It might therefore be expected that hypoglycæmia and hyperglycæmia would be attended by the same, if any, alterations in the curves of the electrocardiogram. This is seen to be the case. The studies of Stepp and Parade, Weichmann, and of Middleton and Oatway, to which reference has already been made, have demonstrated the depression of the T-waves in hypoglycæmia. The present observations recorded above show that in severe untreated diabetes there is also a flattening of the T-waves which become larger as treatment progresses. The effect of insulin in flattening the T-waves in the electrocardiogram has been already referred to in connexion with the present study. Several patients showed an initial increase in the amplitude of the T-waves, which was followed after some weeks of insulin therapy by a reversion to the lesser amplitude (Fig. 5).

SUMMARY.

1. Successive electrocardiograms were taken during treatment in 20 diabetics of all ages. These were at first at intervals of a few days, and later of weeks and months.

2. The tabulated results showed that the most pronounced changes occurred in patients below the age of 40, the T-waves being commonly flattened in leads I. and II., and often diphasic in lead III. Increasing amplitude of the T-waves in leads I. and II., and the substitution of an upright for a diphasic T-wave in lead III. were the effects most frequently observed during treatment. In two patients inversion of the T-wave in all leads was recorded at the outset. All of these changes disappeared progressively during treatment, whether by diet alone or by diet and insulin. The most uniform change and restoration was observed in lead II., while the T-wave in lead III. was more easily influenced by treatment and less stable in its form and direction.

3. Other occasional electrocardiographic changes occurring during treatment were progressive diminution of the P-wave and progressive lessening of slurring in the Q R S complexes.

4. The causal factors underlying the changes described were considered under the following structural and metabolic heads: (*a*) coronary sclerosis; (*b*) coronary intimal fatty deposition; (*c*) abnormal metabolic products; (*d*) defective nutrition of the myocardium.

5. It was concluded that the electrocardiographic changes produced by diabetes represent mainly a parenchymatous damage to the heart resulting from defective nutrition. Coronary disease, whether sclerotic and permanent or of an early reversible fatty type, probably plays a subsidiary rôle in the production of abnormal electrocardiograms.

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WOLVERHAMPTON CONTRIBUTORY SCHEME.—The contributory scheme of Wolverhampton Royal Hospital, which came into operation in October last, is already an assured success. It has been joined by 790 firms within its orbit, and in addition to the work of many district committees, 1400 persons are paying their contributions direct. The 3000 miners employed by the West Cannock collieries have decided to join.

JERUSALEM OPHTHALMIC HOSPITAL.—The ophthalmic hospital in Jerusalem, maintained by the British Realm of the Order of St. John, is being connected by a subway with the recently opened annexe. The annexe is lower down the main road between Jerusalem and Bethlehem than the main building, and traffic has increased so greatly that patients can no longer pass in safety from the one to the other. The work is now approaching completion.

STAPHYLOCOCCAL TOXIN.

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THE hæmolytic and leucocidal actions of staphylococcal toxin have been recognised for many years. Denys and Van de Velde [1] writing in 1895 described the destructive effect upon the leucocytes in the pleural cavities of rabbits following the injection of staphylococci, as well as the appearance of anti-leucocidin in the serum of immunised rabbits. The hæmolytic action of the cocci on blood-plates was noted by Kraus [2] in 1900, and in 1922 an important paper on the subject was published by Julianelle,[3] who demonstrated that the hæmolytic titre did not correspond either to pathogenicity or to leucocidin production. More recently Burnet [4] has added to our knowledge of the toxins and in particular to their mode of production in vitro. Parker Weld and Gunther [5] have described methods of separating the different components of the toxin.

The present paper deals with an investigation, both quantitative and qualitative, of the toxins obtained from 22 strains of staphylococci derived from a variety of human lesions. Our object has been to examine the relative importance of the different toxin constituents in human pathology.

The toxic properties examined were the following: hæmolytic, leucocidal, necrosing, and lethal.

TECHNIQUE AND RESULTS.

1. *Toxin preparation.*—This was obtained by Burnet's method and as previously described by us.[6] After final sterilisation through a Seitz filter the pH was brought to 7·0–7·2, a few drops of chloroform were added, and the surface of the filtrate covered with a layer of sterile liquid paraffin.

2. *Hæmolysin tests.*—Three tubes were put up for each filtrate, giving final dilutions of 1 : 50, 1 : 200, and 1 : 800. Washed rabbit cells were used in final 1 per cent. dilution. After mixing, the tubes were left in the incubator for one hour, reshaken, left on ice over night and read next morning.

3. *Necrosis of rabbit's skin.*—Intradermal doses of 0·2 c.cm. were given. Strongly hæmolytic filtrates used in 1 : 20 dilution commonly gave an area of necrosis 1·5 to 2 cm. in diameter, and this was taken to indicate a strong necrotoxin. Feebly hæmolytic filtrates were used in 1 : 5 and 1 : 20 dilutions, the former being usually feebly positive and the latter negative, indicating a weak necrotoxin. When the 1 : 5 dilution was negative a subsequent test was made with undiluted toxin. Fresh rabbits were always taken and up to six filtrates were tested simultaneously on the same animal. We believe that the results indicate adequately whether a weak or strong necrotoxin was present.

4. *Lethal action.*—This was investigated with four toxins only, Nos. 2, 3, 4, and 6. Young rabbits each weighing 1 kg. were used and a preliminary injection of 0·5 c.cm. was made into a vein. If death did not occur within a short period, further injections were made.

5. *Leucocidin.*—Human blood-cells obtained by venepuncture were washed in saline, a narrow centrifuge tube being used for the final washing in order to concentrate the leucocytes. The toxin, adjusted if necessary to pH 7–7·2, was diluted with saline to 1 : 2 and 1 : 8. Equal volumes of cells and filtrate were incubated for one hour in a Wright's pipette. The contents, after a thorough washing in and out of the pipette, were mixed in the proportions of 2 volumes of the toxin cell mixture with 1 volume of fresh human serum and 1 volume of a staphylococcal suspension. After 20 minutes' incubation the contents were again washed out, the drop thoroughly stirred with a platinum loop, and a film made from a loopful of the mixture and stained with Leishman. The addition of the phagocytic mixture was continued because it provided an additional functional test for the phagocytes and because it was found that this extra dilution of the cells led to more satisfactory films. The use of the platinum loop was adopted because the neutrophils tended to form small clumps, and it was felt that the loop would demonstrate the presence of these clumps more certainly than the usual method of drawing out a blood-film. With some filtrate mixtures after incubation, small masses tending to block the pipette were observed. These clots were caught whenever possible and rubbed out into films for examination.

In the control films many phagocytes were present, often in clumps. With a strong leucocidin (marked A in the Table) all the phagocytes were destroyed in both the dilutions 1 : 4 and 1 : 16 which were employed for the routine test. Well-stained lymphocytes were present. Such toxins usually produced clots, films of which showed masses of almost homogeneous basophil cell debris entangling normal lymphocytes. If this phenomenon occurs in vivo, it supplies a possible explanation of the septic emboli so commonly associated with staphylococcal septicæmia. Filtrates marked C in the Table appear to have no destructive action on the leucocytes in either dilution. B indicates a weak leucocidin with partial or almost complete destruction at 1 : 4 and very little action at 1 : 16. B filtrates sometimes produced clots which were found to contain many intact polymorphonuclears. The red cells, except in the case of one toxin, No. 7, were unaffected.

The Table indicates the amount of each type of toxin in the various filtrates: A represents a strong toxin; B a feeble one; and C almost complete absence of the toxin. In the fourth column an attempt is made to indicate in a similar manner the severity of the human lesion from which the strain of cocci was obtained: A represents a severe lesion such as osteomyelitis or large carbuncle, and A+ a case in which there was evidence of pyæmia; B indicates a small local lesion such as a boil or stye; and C a purely superficial infection such as sycosis barbæ.

Strain.	Hæmolysin.	Necrotoxin.	Leucocidin.	—	Human lesion.			—
					Sex.	Age.	D.*	
1	A	A	A	B	M.	32	–	Boil on buttock.
2	A	A	B	C	M.	30	–	Sycosis barbæ.
3	B	B	A	A	F.	46	+	Severe carbuncle of neck.
4	B	B	A	A+	F.	13	–	Fatal infection of lip.
5	C	C	C	A	M.	4	–	Osteomyelitis of tibia.
6	A	A	C	C	M.	2	–	Skin eruption on face.
7	A	A	C	B	M.	52		Prostatitis.
8	A	A	A	B	M.	49	–	Small carbuncle of wrist.
9	B	B	A	A	M.	21	–	Severe carbuncle of neck.
10D	A	A	B	C	M.	35	–	Sycosis barbæ of 15 years' history.
10P	C	C	C	C				
11	B	B	A	A+	M.	17	–	Three years' pyæmia following bone infection.
12	A	A	B	A	F.	55	+	Carbuncle: septicæmia two years before.
13	B	B	B	B	F.	19	–	Severe infected hand.
14	B	B	A	A+	F.	15	–	Osteomyelitis of humerus and empyema.
15	A	A	C	A	M.	5	–	Acute osteomyelitis with rapid improvement.
16	A	A	A	B	M.	19	+	Nasal furuncle following many small infections.
17	B	A	A	B	F.	46	+	Stye following carbuncle in Case 3.
18	A	A	C	C	F.	28	–	Sycotic condition on leg.
19	B	B	B	A	M.	13	–	Empyema following wound of thorax.
20	A	A	B	A	M.	65	+	Severe carbuncle.
21	A	A	B	A	M.	14 / 365	–	Fatal abscess of back, possibly after injury.
22	B	A	A	A+	F.	21	–	Fatal infection of lip.

D* = diabetes.

Analysis of the Table shows that the hæmolysin and necrotoxin vary together on the whole, but that slight discrepancies may occur, as with Nos. 17 and 22. Both of these gave weak hæmolysin, but the necrotoxin had to be labelled A, though the reactions were definitely feebler than the majority in this class. These slight differences tend to support Parker Weld and Gunther in their contention that the hæmolysin and necrotoxin are distinct.

FOUR GROUPS OF STRAINS.

Comparison of the hæmolysin and leucocidin permits a division of the strains investigated into four groups:—

1. Those with high leucocidin and low hæmolysin.
2. Those with high leucocidin and high hæmolysin.
3. Those with low leucocidin and high hæmolysin.
4. Those with low leucocidin and low hæmolysin.

It is advantageous to compare these groups with the types of human lesions from which the strains were derived.

Group 1 consists of seven strains (Nos. 3, 4, 9, 11, 14, 17, 22) with strong leucocidin and weak hæmolysin. All these strains were obtained from severe lesions, the only exception being No. 17, which was grown from a small stye, following a carbuncle from which No. 3 was obtained, and it is to be presumed that the two strains were identical. All the four strains from pyæmic cases fall in this group and include two strains from rapidly fatal cases of carbuncle of the lip. The association therefore of this class of toxin with virulent disease in man is remarkably constant.

Group 2 comprises three strains (Nos. 1, 8, 16) rich both in leucocidin and hæmolysin. Two were derived from a common, small single boil, and one from a small carbuncle, of short duration, which yielded rapidly to treatment. The few strains of this group in our Table may be due to the fact that these were the only cultures examined from this common and perhaps most typical staphylococcal lesion in man.

Group 3.—Strains with weak leucocidin and strong hæmolysin (Nos. 2, 6, 7, 10D, 12, 15, 18, 20, 21). These strains were obtained from lesions of very different severity but some classification is possible. The most definite finding is that all the sycosis cases fall within this group—namely, 2, 6, 10D, 18. No. 7 may well be a secondary infection; No. 15 came from a case of acute osteomyelitis which cleared up rapidly after operation and without complication. No. 21 was obtained from a baby of 14 days with a strong suspicion of injury and presumably poor resistance. Nos. 12 and 20 both came from carbuncles in elderly diabetics.

Group 4.—Strains weak both in leucocidin and hæmolysin (Nos. 5, 10P, 15, 19) have some clinical evidence of a saprophytic origin; 10P grew in pale colonies and was isolated from strain 10D which yielded a strong hæmolysin. Nos. 15 and 19 both came from infected wounds, one of the hand, the other of the thorax. No. 5 strain is unique in our experience in that it produces no hæmolysin at all and no detectable leucocidin. It was derived from an osteomyelitis lesion in a child of 3 years. It is difficult to account for the appearance of this strain and its inclusion in this group. A streptococcus was subsequently isolated from the pus of the case, but the staphylococcus was obtained twice from earlier specimens in apparently pure culture.

The lethal action for rabbits was tested in the case of four toxins only. Nos. 6 and 2 were chosen as being strongly hæmolytic and feebly leucocidal, Nos. 4 and 3 as being of the reverse order; 0·5 c.cm. of the hæmolytic toxin No. 6 was given to two rabbits, the first died in three minutes, the second showed symptoms, but recovered and was given a further 1 c.cm. two hours later. Death was almost instantaneous. The other hæmolytic toxin No. 2 killed a rabbit in two minutes. Leucocidal toxin No. 4 was given to two rabbits in doses of 0·5 c.cm., followed two hours later by 1 c.cm. and 30 minutes later by a second 1 c.cm., without effect. Similarly toxin No. 3 showed no lethal action.

It appears therefore that the lethal action of a toxin for rabbits runs parallel with the necrosing effect on the rabbit's skin and the hæmolytic titre for rabbit red cells, but is distinct from the leucocidal action on human white cells.

Cultural characters.—The separation of 22 strains of staphylococci into four groups on the basis of toxin production suggested the possibility that cultural differences might be detected for the groups. The full cultural characters of 16 strains were investigated. In the fermentation reactions of the 13 carbohydrates selected all showed complete uniformity. A few strains acidified but failed to clot milk, and three strains (Nos. 7, 8, and 18) failed to liquefy gelatin. There was thus no association between cultural characters and toxin production. The peculiar strain No. 5 which yielded no toxin gave typical cultural reactions throughout.

DISCUSSION.

Antitoxin production.—In a recent paper with Mr. V. W. Dix the use of an antitoxic serum prepared for us by Dr. G. F. Petrie was described. This serum was derived from a horse inoculated with a single strain toxin of high hæmolytic titre. It was shown to have the power of neutralising both the hæmolytic and necrosing properties of the toxin. The toxin was that derived from strain No. 1 in the Table, and was subsequently found to have a high leucocidal titre also. Titration of the concentrated antiserum against normal horse serum showed an anti-leucotoxic power of 20 to 1. The selection of this toxin for immunisation was thus, in part, a matter of chance. It is, at least, suggested that the most important toxic fraction in severe human lesions is the leucocidal, and it is possible that a more potent serum might be obtained from the use of a Group 1 rather than a Group 2 toxin.

In our description of the toxic action upon the leucocytes we have suggested that the tough macroscopic clots found in vitro may be a source of pyæmic emboli in vivo. Dr. Petrie's antitoxic serum seemed most efficacious in the pyæmic cases and we noted at the time the complete cessation of metastatic abscess in all the cases so treated.

Constancy of toxin production.—The 22 staphylococcal strains have been divided into groups on the basis of the proportional production of hæmolysin, with which are associated necrotoxin and lethal toxin, and of leucocidin. Only four main divisions are possible and all are represented in our strains. It can be said therefore that the staphylococcus may put out much or little hæmolysin or leucotoxin or both, and any classification on these lines would be without meaning unless these outputs are constant for the strain. We believe that toxin production by the strain is constant within such limits as we have been able to examine them. The majority of these strains have been isolated for some months, from many of them fresh toxins have been periodically prepared on different occasions and no serious alteration in the

relative proportion of hæmolysin to leucocidin has ever been proved. The peculiar strain, No. 5, which grows in unusually small colonies on agar (though in its general cultural properties identical with the other strains) was twice isolated from the same patient. The Group 1 strain No. 3 isolated from a carbuncle was again obtained from a stye in the same patient four months later. The association of certain classes of toxin producers with certain types of human lesions is, we think, as close as could reasonably be expected in view of the comparative crudity of our measurements and of variations in the resistance of the individuals affected, and such association supports the belief that toxin production is a constant, at any rate for the period of infection. Clinical instances also occur to us of the accidental transfer of similar staphylococcal lesions from one person to another, for example, a nursemaid with a stye transferring to a child an identical lesion or a small superficial boil.

Toxin and cell species.—A survey of the literature shows that, in estimating toxic action, the animal species from which the test cell was derived seems to have been considered a matter of indifference. Rabbit red and white cells were employed by Neisser [7] and Wechsberg, and subsequently by others, while Julianelle [3] compares the lethal action on mice or rabbits, the hæmolytic titre for horse red cells, and the leucocidal action upon guinea-pig cells. We find that toxins with a high titre for rabbit cells commonly have a lower titre for sheep cells, and may have no action upon human cells. The titre for rabbit cells often reaches 1 in 800, that for human cells rarely attains 1 in 5. Previous saturation of a toxin strongly hæmolytic for rabbit and inactive for human red cells with human cells does not reduce the rabbit cell titre. It is true that a clear zone often appears around staphylococcal colonies on human blood-agar plates, but only one of our 22 strains yielded a titre for human cells of any strength. This toxin (No. 7) gave incomplete hæmolysis throughout with a trace up to 1 in 800 and saturation of this toxin with human cells did not seem to affect the subsequent titre for rabbit red cells. It appears that human and rabbit red cell hæmolysins are distinct, and that staphylococcal toxin rarely acts upon the human red cell in vitro. The leucocidin was tested against human cells, but in some instances was tried against rabbit white cells also. It is difficult to compare these results; the human cell experiments were clear-cut, the rabbit cell results may have been prejudiced by the hæmolysis which was nearly always present, and by what appeared to be the necrosing action of the toxin imposed upon the leucocidal action. It appeared that toxin leucocidal for human cells destroyed rabbit cells also, but toxins with low titres for human cells had considerable action upon rabbit cells. There was some histological suggestion that the main effect of the latter toxins was rather upon the cell membrane than a disruptive action upon both nucleus and cytoplasm as would seem to be the case with the Group 1 toxins.

TREATMENT.

The association of certain combinations of toxins with certain types of staphylococcal infection suggests differences in the specific treatment of these conditions. We have found the employment of an antiserum to be chiefly efficacious against pyæmic infections and have already indicated our belief that the important antibody in the serum is probably the anti-leucocidin. The sycosis cases were, on the contrary, all associated with a strong hæmolysin and a weak leucocidin. These cases notoriously respond badly to vaccine treatment and are sometimes made worse by it. There is also some suggestion that they do worse with an autogenous than with a stock vaccine. In these infections the hæmolysin may be associated with the sensitising factor to which we have previously drawn attention.[8] It appears to us that treatment with toxoid in these and possibly in other staphylococcal lesions is more rational than treatment by vaccine, and that in the sycosis cases the toxoid should contain leucocidin and possibly the minimum amount of hæmolysin. We have treated only two such cases but both were carefully selected as examples of extreme chronicity, widespread lesions, and resistance to every form of treatment. The result in both cases has been sufficiently encouraging to test the method further. Immunisation by toxin or toxoid is, in theory, preferable to vaccine therapy. The vaccine dose comprises a roughly calculated number of dead bacteria, and a very uncertain amount of toxins, mixed in quite unknown proportions. The dose of toxoid can be accurately measured and by a choice of toxin the relative proportions of the different constituents can be chosen at will. The comparative values of toxin and vaccine treatment can only be determined by thorough clinical trial.

SUMMARY.

(1) Toxic filtrates have been prepared from 22 strains of *Staphylococcus aureus*. Their capacity to hæmolyse rabbit red cells, necrose rabbit's skin, and destroy the leucocytes in human blood has been estimated. (2) The results confirm the findings of others, that the hæmolysin and necrotoxin vary closely together with no relation to the leucocidin. (3) The lethal action for rabbits, when investigated, was found to vary with the hæmolysin content of the filtrate. (4) Comparison with the human lesions indicates that strains yielding strong leucocidin and weak hæmolysin are commonly associated with acute severe infections and the reverse combination with long-standing superficial infection of the sycotic type. (5) Up to the present no evidence of variation in the qualitative production of toxins by the strains has been obtained. (6) Suggestions as to possible lines of treatment are discussed.

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LUNACY IN THE IRISH FREE STATE.—According to the inspection report submitted to the Grangegorman Mental Hospital committee there is a continuous increase in the number of persons in the Free State of unsound mind; the two institutions of Grangegorman and Portrane contain 3750 patients from the counties of Dublin, Louth, and Wicklow. The report continues: "Statistics for the Free State show that one in every 200 of the population is a patient of a public mental asylum. In addition, there are a large number in private asylums, workhouses, and hospitals, and a large number are hidden away in private homes. Add to this the number of nurses, attendants, and other officials engaged in the work of these asylums, and you will approach closely to a figure of one in every 100 of the population not only dead to the State, but actually a burden to the State. A figure of one in every 300 would be a more normal level." Bad housing is suggested as one of the causes of mental disorder.

HÆMORRHAGIC PURPURA FOLLOWING SCARLET FEVER.

REPORT OF TWO CASES IN ONE FAMILY.

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A small outbreak of scarlet fever began in Oxford about Feb. 8th, 1931, seven cases being reported, four of which came under the observation of one of us (F. G. H.). All the cases were mild, and the only common factor was that all received their milk from the same dairy. A careful investigation by the public health authorities threw no light on the source of infection. Of the seven cases, four had been receiving Grade A milk supplied by the dairy, while three had received the ordinary milk, the two supplies being kept quite distinct. With the exception of the two cases which form the subject of this report, all made an entirely uneventful recovery and convalescence.

CLINICAL NOTES (F. G. H.).

The cases with which this account is concerned appear to us to be unique, so that it is wise to mention all details which may be relevant.

Family history.—The parents of the children concerned are distant cousins. On neither side is there any history of "bleeding" or other morbid inheritance, and longevity is the normal expectation of the members of both families.

Past history.—The three children in this family, a girl aged 7, a boy aged 5, and a girl aged 3, were all healthy children who had hitherto experienced only minor ailments and escaped any of the specific fevers. All three were inclined to have large tonsils and adenoids. They had unusually liberal allowances of fresh food in the form of fruit, salads, milk, eggs, and wholemeal bread, and spent much time in the open air. All were dark-haired and well nourished, with dark eyes and high-coloured rosy cheeks.

History of the cases.—On Feb. 8th the boy, aged 5, contracted scarlet fever, the onset being marked by fever and a sore-throat. All three children had been playing together on the 8th, and the exposure of the two sisters was obvious. The patient and his elder sister had three years previously been given prophylactic diphtheria antitoxin; therefore no anti-scarlatinal serum was administered in either case for fear of a serum reaction. The younger sister was given a prophylactic dose of anti-scarlatinal serum, and protected. On Feb. 12th the elder sister, aged 7, developed scarlet fever. The two children were then nursed in the same room, the beds being at least eight feet apart. In each case the scarlet fever was of a mild order, the temperature lasting only three days, and in neither case reaching 103° F. at its highest. The initial fever subsided, but both children simultaneously developed an acute bronchitis, introduced by an attendant, and were febrile for a further period of three or four days. There was at the same time some enlargement of the glands under the jaw from the infected tonsils.

On Feb. 23rd, 16 days from the onset of the scarlet fever, the first patient developed hæmorrhagic purpura and was dead in 48 hours, the temperature having been normal for the previous five days and the child's general condition excellent. On March 5th, 21 days from the onset of the scarlet fever, the second patient developed hæmorrhagic purpura and was dead in three days, the temperature having been normal for the previous seven days and the child's general condition excellent.

CLINICAL AND PATHOLOGICAL NOTES.

Case 1.—The boy, when first seen on Feb. 23rd, 16 days from the onset, was desperately ill, waxy yellow in colour, with a barely detectable pulse at the wrist, and having vomited normal stomach contents. His temperature was 98° F. There was a hæmorrhage under the skin on the left cheek and extensive hæmorrhages under the skin of both elbows, with a number of pin-point hæmorrhages over the abdomen and some more extensive hæmorrhages over the buttocks and backs of the calves. Albumin was abundant in the urine. A blood count at this time showed:—

Red cells	4,890,000	Polymorph	71%
White cells ..	27,250	Lymphocytes	21%
Platelets in large numbers.		Endothelial	8%

Adrenalin, ♏ ii., was given four-hourly, and the pulse-rate varied between 100 and 120 during the first 24 hours, the temperature rising to 99° F. in the evening, and fluid nourishment being well taken during the day. After a restless night, with perpetual complaints of pain in the limbs, there was a rapid advance of the hæmorrhages in all areas where they were previously present; further hæmorrhages developed in the right cheek, and the left ankle-joint became distended with blood.

The waxy yellow colour of the skin of the whole body persisted throughout, contrasting vividly with the deep purple-black in the areas of the hæmorrhages. The child died during the early hours of the second night, with all the appearances of death from hæmorrhage: cold, sweating, sighing respiration, and a failing, increasingly rapid pulse. At no time was blood present in the urine, stools, or vomits; the subcutaneous hæmorrhages were, in the main, present on pressure areas. No post-mortem examination was made of this case.

Case 2.—The girl, when first seen on March 5th, 21 days from the onset, presented a strikingly distinctive picture. Both calves, from a sharply defined and symmetrical line at the junction of the middle and upper third of the calf downwards, were deeply cyanosed, though warm, while multiple small hæmorrhages were visible in the deep blue skin on the dorsal aspect of each foot. A few pin-point hæmorrhages were also visible over the lower abdomen. Scattered over the posterior surface of the upper arm on both sides and along the outer side of each thigh were white areas of skin, varying from a threepenny-piece to a shilling in size, surrounded by an area of hyperæmia some few millimetres wide.

The temperature was normal, pulse-rate 100, and the general condition of the child good, although vomiting had occurred, as with the brother. The child was a little paler than normal, although there was at this time no sign of the waxy yellow pallor of the previous case. There was abundant albumin in the urine, although it had been absent up to the previous day, daily specimens having been tested during the preceding week. In the course of the next three hours the "blue boots" appearance entirely disappeared, as did the patches on the arms and thighs which had been surrounded by zones of hyperæmia.

Adrenalin, ♏ ii., was administered four-hourly, and liquid liver extract was given by mouth.

Immediate steps were taken to examine the blood with a view to the possibility of a transfusion. This examination revealed a most unusual condition:—

1. The patient belonged to blood group 2.
2. The patient's father was also group 2, the mother being group 4, but the patient's serum agglutinated the cells of both parents.
3. The patient's serum also agglutinated the corpuscles of her sister, who was also blood group 2.
4. The patient's serum also agglutinated the cells of five group 2 donors, and 12 group 4 donors.
5. The patient's serum agglutinated a suspension of her own red cells.

In consultation with Dr. H. Letheby Tidy, it was agreed (*a*) that blood transfusion was impossible, and (*b*) that the administration of serum, after proper precautions to avoid anaphylaxis, might be worthy

of consideration, and that anti-scarlatinal serum was probably the serum indicated.

During the second day the hæmorrhages rapidly developed, areas of hæmorrhage appearing on the extensor surfaces of both forearms and buttocks, while the left calf became distended with subcutaneous hæmorrhage to about twice its normal size. Late in the day it was necessary to administer morphia to relieve pain. By the evening of the second day the child was beginning to develop the waxy pallor noted in the first case. Five minims of anti-scarlatinal serum were given subcutaneously without any consequent reaction, and one hour later 5 c.cm. was administered intramuscularly.

The hæmorrhages remained constant, but intense thirst, accompanied by persistent vomiting of all fluid taken by mouth, continued during the night. Rectal salines were given and retained, but the child died during the morning of the third day, the final picture being, as in the case of the first child, death from hæmorrhage.

The total amount of blood extravasated in the different lesions of the second child was small in quantity, and not in itself adequate to account for death.

POST-MORTEM INVESTIGATIONS (A. G. G.).

In Case 2 autopsy was performed 48 hours after death.

The body was moderately nourished. The greater part of the skin of both legs below the calf was purple and ecchymotic, and there were large blebs containing blood in the left leg. There were patches of purpura on the buttocks and on the extensor surfaces of both hands and wrists, and smaller petechial hæmorrhages in the upper arms. There were 2–3 c.cm. of clear fluid in the pericardium; otherwise the cavities were dry and natural. There was a local hæmorrhage in the fat of the anterior mediastinum near the pericardium.

Tonsils and lymph glands.—Both tonsils were large, pale, and on section extremely cellular, showing no crypts except one near the surface of the left tonsil which exuded mucoid, opaque fluid. There were no hæmorrhages. The neck glands below the tonsils were slightly enlarged, and on section showed the same highly cellular appearance. The tongue, pharynx, œsophagus, trachea, thyroid, and aorta were normal. The glands at the bifurcation of the trachea were small, fleshy, and dark red.

Thorax.—The heart seemed natural, with no sign of hæmorrhage; the muscle was on the pale side, but of good texture and colour. Both lungs showed moderate hypostasis, and there were a few minute petechiæ underneath the pleura. They were otherwise dry and showed no change.

Abdomen.—The mucous membrane of the stomach and intestines was normal. The liver was slightly brownish, firm, and showed the lobules under a low magnification. There were no visible hæmorrhages. The spleen was not enlarged. There was a hæmorrhage on the convex surface towards the upper pole: it had not spread for any distance into the pulp. The pulp was firm, Malpighian bodies were easily seen, and most of them had a zone of hyperæmia surrounding them. The suprarenals were thin, and the pigment layer in the lowest part of the cortex was evident. Both kidneys were pale and showed no abnormal change. The bone-marrow of the ribs was slightly paler than normal, otherwise without change. Incision into the hæmorrhage in the right leg showed that it was entirely superficial to the periosteum of the tibia, which was normal in appearance.

MICROSCOPICAL APPEARANCES.

Left tonsil.—Very highly cellular and cannot be recognised as a tonsil under the low power. Crypts that can be recognised under the high power are small cavities lined by a single layer of epithelium. The sites of other crypts are probably represented by areas in which with some epithelial and lymph cells there are numerous small globular bodies, possibly from nuclear necrosis. No myeloid metaplasia. The tonsillar glands show extreme lymphoid hyperplasia.

Right tonsil.—The crypts can be distinguished, and while the walls of some are infiltrated with lymphocytes some are normal, but none contains pus. There are several large fibrous strands in this tonsil infiltrated with lymphocytes. Areas of necrosis are less evident than in the left.

Spleen.—Hæmorrhage surrounds some of the Malpighian bodies. Some of the sinusoids are plugged with hyaline clot. A section of the hæmorrhage showed the tissue turgid with blood. Malpighian corpuscles small. Veins have more white cells than normal. No hyperplasia.

Thymus.—Lymphatic tissue normal. Of Hassal's corpuscles some are normal, some distended with epithelial debris. No suggestion of infection.

Bronchial gland.—Hyperplasia with patches of (?) necrosis as in tonsil. Hyperæmia. Increase of white cells in some vessels.

Mesenteric gland.—Hyperplasia.

Bone-marrow of ribs.—Packed with neutrophil and a few eosinophil myelocytes. Normoblasts present.

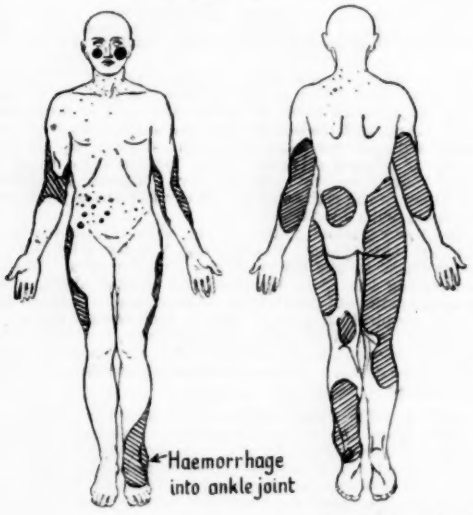
Mediastinal hæmorrhage.—Some parts appear without more than a normal number of white cells. Others contain an excess. Two small vessels are packed with white cells.

Skin.—The hæmorrhage here does not contain a large number of white cells (cf. the splenic and mediastinal hæmorrhages). No reaction of tissue or blood-cells in relation to hæmorrhages from capillaries.

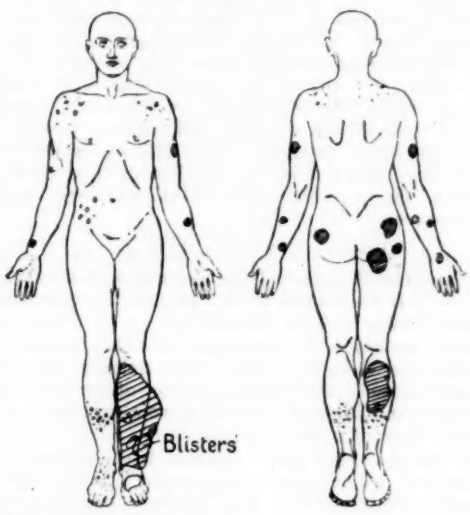
Heart.—Very little blood seen in the vessels; the arterioles were empty and collapsed.

Kidney.—Practically normal. No signs of nephritis. A few blood-cells in glomerular capsule. Capillaries empty

CASE 1.



CASE 2.



Distribution of purpura in the two cases.

or containing no excess of white blood-cells. Larger veins distended and contain a large proportion of white cells.

Liver.—Cellularity along Glisson's capsules.

Blood from Right Ventricle (Differential Count).

Myelocytes	28·2	68·0	....	141
Polymorphs	4·0		....	20
Transitionals	35·8		....	179
Small lymphocytes	26·2	30·8	....	131
Large "	4·6		....	23
Endothelials	0·4		....	2
Myeloblasts	0·6		....	3
Eosinophil myelocytes	0·2		....	1
	100·0		....	500

COMMENTS.

The unusual features of the two cases here reported may perhaps be usefully enumerated.

(1) Purpura hæmorrhagica as a late sequela of scarlet fever appears to be extremely rare. (2) This complication developed in two children of the same family, 16 and 21 days respectively after an attack of scarlet fever which clinically was of a mild type and unaccompanied by any evidence of unusual toxæmia. (3) While the mode of death was characteristic of death from hæmorrhage, the actual loss of blood evident in the one case subjected to post-mortem examination was small in comparison with the clinical anæmia. (4) In contrast with the case reported by Box and Massingham, there was no reduction in the platelets. (5) It would seem possible to conclude only that in these children there existed some unusual constitutional susceptibility to the scarlatinal toxin.

While the present paper was in preparation, Dr. F. G. Wood-Smith kindly allowed us to see the report of a similar case occurring in an adult, and with his permission the bibliography is appended.

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CLINICAL AND LABORATORY NOTES

A NOTE ON CHRONIC INFECTIONS OF THE LUNGS.

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NUMEROUS papers have been published describing chronic infection of the lung by fungi, and the existence of streptothrix infections of the lungs is well established as a clinical and pathological entity.[1] The weight of evidence against the view that monilia are non-pathogenic to man is rapidly increasing.[2-8 10] It is hardly conceivable that an organism should exert a pathogenic effect when acting alone and yet be looked upon as non-pathogenic when associated with known pathogenic organisms.

Since 1922 work has been carried out in Jersey on the differential diagnosis of chronic infections of the lungs. The unsatisfactory diagnosis of "closed T.B." led to the hope of finding an organism which might be simulating infection with the tubercle bacillus. With this in view all specimens of sputum were examined, both microscopically and culturally.

The cultural examinations brought to light the presence of blastomycetes, chiefly monilias. Where, on clinical grounds, tubercle had been suspected, but the bacillus had not been found, monilias were present. Monilias were also present in tuberculous infections, and the clinical difference between the mixed infection and the rare condition of a simple tuberculous infection was marked, the mixed infection being an acute condition whilst the simple one was chronic, and more resembled a carrier state.

The prevention of secondary infection with pyogenic organisms is the key to the brilliant results obtained in the modern treatment of surgical tuberculosis. The contrast between surgical and medical tuberculosis lies in the fact that the latter, when seen by the physician, is always complicated by a pyogenic infection, chief of which are monilias. Where tubercle of the lung is a primary and single infection, it does not require treatment, and recovery is natural. This may be a somewhat startling suggestion, but the following further facts are put forward in its support.

The history of a tuberculous patient often includes an attack of a zymotic disease such as whooping-cough or measles, which has left a legacy of bronchitis or broncho-pneumonia. Many cases give a history of pleurisy, and monilias have been recovered from pleural effusions. I have known cases from which monilias have been grown out, and which within a year or so—not having been treated—have developed a secondary infection of tubercle. Chevalier Jackson,[3] using the bronchoscope, has recovered monilias direct from infected lung tissue. N. Hamilton Fairley,[7] in a discussion on a paper by Castellani, refers to the infiltration of lung tissue with gelatinous masses, in which monilia are found in large numbers.

The following Table gives the extent and the results of the work in Jersey during the four years 1927–30.

Year	1927	1928	1929	1930
Total number of patients examined ..	295	304	376	381
Number due to T.B., and blastomycetes	80	72	92	79
Number due to blastomycetes only ..	120	160	189	247
Number due to other organisms ..	95	62	94	55

Preventive treatment against infection with monilias consists in the pasteurisation of milk. Cows suffering from mastitis excrete monilias in their milk. Warts on the teats are an indication of the infection.[9]

The keynote to the treatment of chronic infections of the lungs is the removal of pyogenic organisms by drainage. Free drainage brings about improvement, with fall of temperature, clearing-up of the sputum,

and increase in weight. Any recurrence of symptoms is found to be due to a relapse of the monilia infection. Pneumothorax and aspiration by the bronchoscope are simply surgical methods of drainage, but such treatment is applicable to gross lesions. Medical treatment requires the institution of medical drainage at the earliest possible moment. When sputum is tenacious, there can be no free flow of secretion. When a thick purulent or gelatinous sputum is present, culture will usually reveal the presence of monilias, and on the finding of monilias, medical drainage must be commenced.

Alkaline potassium iodide is the drug for the purpose; it acts in the following ways: (*a*) sputum is liquefied; (*b*) a free flow of secretion is set up from within outwards, thus preventing the ingress of organisms; and (*c*) the drug has a selective action on fungi. It has been stated that iodides tend to decalcify the body. What effect has chronic suppuration on the calcium content of the body? If iodides do decalcify, then the answer is: replace the calcium loss by the exhibition of a calcium salt.

Vaccines are used to increase immunity against monilia. The vaccine is used in two forms. A first course consists of monilia grown on agar; this only contains the yeast form. When monilia persists after a first course, a second course is given where the monilia is grown on Sabouraud; the vaccine thus grown contains all elements of monilia, including mycelium. Tubercle vaccine is only used when the patient is apyretic. If acute symptoms return a cultural examination of the sputum is made, and if monilia is found, the patient reverts to monilia vaccine.

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RUPTURE OF AN ANEURYSM OF THE ABDOMINAL AORTA.

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A WOMAN, aged 68, admitted to King's College Hospital, said she had suffered for a number of years from what she called rheumatism. But the illness which brought her into hospital started only three weeks prior to admission, before which she had been comparatively healthy.

She complained of pain in the right lumbar region. It was never very acute, but a dull heavy ache, localised in the right loin. She began to feel very ill, complaining of nausea and loss of appetite at first, and later she began to vomit. For a few days before admission she had been unable to keep any food down at all. She felt very thirsty but she vomited even water. Hiccup started three days previously, and had been persistent up to the time of admission. She had gradually become more drowsy and apathetic. The bowels had been very constipated; hæmaturia, which had been noticed for a week, had persisted, but there had been no frequency or pain on micturition. The illness had started with a febrile attack, and the temperature had remained elevated for the whole three weeks prior to admission. Before the hæmaturia started there was no albumin or pus in the urine.

When the patient was first seen, she presented a typical picture of uræmia. She was very drowsy, could only be roused with difficulty, and talked incoherently. The tongue was red and very dry. There was persistent hiccup and the patient kept retching and vomiting. She was parched with thirst. The temperature was 100·6°, the pulse-rate 100, and the respiration-rate 22. The pulse was poor in volume, although the blood pressure was 160/90. There was slight twitching of the left eye; the discs were normal. On examination of the abdomen there was found to be a large firm tumour in the region of the left kidney. The swelling was tense and very tender, but was not absolutely fixed. There was a transmitted pulsation.

Examination of a catheter specimen of urine showed the presence of a number of red blood-cells and pus cells. There were no casts, crystals, or epithelial cells seen, and no organisms in direct smears. The culture was proved subsequently to be sterile. The blood-urea was found to be 217 mg. per 100 c.cm.

The diagnosis of a renal tumour causing uræmia seemed reasonable, and the patient was treated accordingly. She was given one pint of saline with 5 per cent. glucose subcutaneously, and this was given again intravenously six hours later. She was given purgatives and diuretics as well. For several hours the general condition improved considerably; the vomiting almost stopped, and the patient was less drowsy and more coherent. Quite suddenly about 12 hours after admission she collapsed and died.

Post-mortem revealed an astonishing and unexpected condition. On opening the peritoneal cavity it was found to contain nearly two pints of free blood. The tumour in the kidney region was an enormous perinephric hæmatoma, about the size of a melon, completely enveloping a small arterio-sclerotic kidney. The right kidney was also a small arterio-sclerotic one. This was apparently the explanation of the raised blood pressure and the undoubted uræmia. The whole of the aorta (thoracic and abdominal) showed marked syphilitic scarring. There was a rupture of the aorta about 1½ in. above the bifurcation of the aorta on the left side, communicating with an aneurysmal sac the size of an orange. Leakage from this had caused the perinephric hæmatoma, and further rupture of this into the peritoneum had caused the terminal hæmorrhage, from which the patient died suddenly. The aortic valves showed early syphilitic lesions. There was a gumma in the liver adjacent to the gall-bladder and adherent to the second part of the duodenum.

This case is recorded by kind permission of Mr. J. Everidge.

WEST SUSSEX CONTRIBUTORY FUND.—Five years' working of this scheme has resulted in a membership of approximately 17,800, and during this period the sum of £16,461 19*s.* 8*d.* has been paid to the associated hospitals for treatment of members; 853 members, or their dependents, benefited as in-patients, and 1447 as out-patients. Of these, 757 and 1407 respectively were treated in associated hospitals, the remainder going to other hospitals in Sussex. These figures are exclusive of children of members treated under the county council for tonsils and adenoids, and dental treatment paid for by the fund. While the committee is satisfied with the steady progression of the fund—there was an increase of just over £400 in income during 1931—it is pointed out in the fifth annual report "that in some outlying districts served by the smaller associated hospitals, the idea is still prevalent that the fund belongs to the Royal West Sussex Hospital, and that local people do not realise that their own hospital is just as much a part of the fund as the larger one." The suggestion is made that if all the associated hospitals took individual steps to identify themselves with the fund in their own localities, this false impression would soon be removed, and result in an immediate increase of membership. A very satisfactory feature of the working of the fund during 1931 was the complete liquidation of a suspense account which was opened at a bank to finance the formation of the fund.

MEDICAL SOCIETIES

HUNTERIAN SOCIETY.

Ætiology of Chronic Bronchitis.

In the course of his Hunterian Oration, delivered at the Apothecaries' Hall to this society on Feb. 25th, Dr. J. B. Christopherson pointed out that chronic bronchitis is prevalent in all walks of life. There is no commoner disorder in England, and there is no more frequent, direct and indirect, cause of disablement and death in the British Isles. Dr. Christopherson's main thesis was that besides the undoubtedly bacterial invasion of the bronchial mucous membrane, there is a more fundamental cause underlying chronic bronchitis, of nervous origin. Its frequent and intimate association with asthma, and the well-established fact that asthma is anatomically due to constriction of the bronchial muscle, had first led him to think that chronic bronchitis might be caused by over-action of the sympathetic which, with the vagus, controls the bronchi. He believed the turgescence of the mucous membrane, in the bronchioles of chronic bronchitis patients, to be an anatomical fact of the greatest importance. This turgescence was recognised by important authorities as an essential part of the asthma syndrome and, according to Dr. Christopherson, it is probably the ultimate cause of death, in the majority of patients, who die of chronic bronchitis and bronchial asthma. He did not concur with the general opinion that the whole pathology of chronic bronchitis is bacterial, and that the turgescence is inflammatory in nature; in his view there exists a non-bacterial turgescence of chronic bronchitis, which owes its cause to the action of the involuntary nervous system. There was not as yet sufficient experimental proof as to whether it was produced by vagal or sympathetic action. He discussed the evidence in favour of regarding chronic bronchitis as (1) a bacterial, (2) a vasomotor disease, and proceeded to consider the clinical evidence suggesting a connexion between chronic bronchitis and the involuntary nervous system, especially over-action of the sympathetic. He pointed out that an asthmatic child admitted to hospital with an acute attack of spasmodic asthma will recover from his asthma in a few hours without relapsing, but the wheezing râles remain though there is no obstruction to air entry. He regarded the asthmatic attack as vagal, and the state following the attack as the reacting sympathetic portion of the vago-sympathetic complex—vascular turgescence and dilatation of bronchial musculature.

Dr. Christopherson thought that the fact that chronic bronchitis frequently dates from an acute disorder of the lungs might possibly find its explanation in the hypothesis that the normal vago-sympathetic balance has been destroyed by the acute illness. The fact that chronic bronchitis and asthma frequently alternate suggested a relationship of physiological balance, the vagal influence dominating at one time, and the sympathetic influence dominating at another. It was an interesting fact that in bronchial asthma the paroxysms occur at night when sympathetic stimuli are in abeyance. Chronic bronchitis was a daytime disease, when sympathetic influences are strongest. Another possible instance of a fluctuating physiological balance was that, during the asthmatic spasm the cough is hard, expectoration scanty; after the spasm, when the sympathetic influences are beginning to dominate, the cough is loose and expectoration increases. He believed hypertrophy of the submucous tissue to be a sympathetic phenomenon; it was the most serious part of the vago-sympathetic complex. In the cases of asthma operated on by sympathectomy, a number of successes had been reported. Spasmodic asthma being a vagal disorder, sympathectomy could only benefit by cutting out any sympathetic phenomena present. Reported successes following operation on the sympathetic nerve, in cases of asthma, might be due to the fact that the operator had attacked and relieved the sympathetic portion of the asthma complex. There appeared to be no recognisable pathological difference post mortem between chronic bronchitis and bronchial asthma. The problem of chronic bronchitis was a complicated one, and it might be that bacterial influences must also be considered, and perhaps linked up with the physiological. It was possible to link the undoubtedly inflammatory forms of bronchitis with the admitted nervous disturbance in asthma, and to account for the numerous cases, which seemed to fall between the two extremes, on the basis of bacterial allergy. If chronic bronchitis was clinically a part of the vago-sympathetic complex of asthma, asthma and bronchitis might profitably be investigated together. The success of adrenalin as a stimulant of the sympathetic nerve encouraged the hope that a rival antagonistic substance—pharmacological or biochemical, or of the acetyl-choline type—might be found to stimulate the bronchial vagus without risk.

ROYAL SOCIETY OF MEDICINE.

SECTION OF COMPARATIVE MEDICINE.

Milk Fever and Similar Conditions.

At a meeting of this section on Wednesday, Feb. 24th, Dr. J. A. Arkwright, F.R.S., the President, being in the chair, a discussion took place on Milk Fever in Parturient Cows and Similar Conditions in Other Animals and the Human.

Prof. J. Russell Greig, in opening, said that the pathology of the condition known as milk fever in cows, like that of eclampsia in women, to which it bore a clinical resemblance, had long been obscure. In most instances it attacked cows within 72 hours after calving, the parturition itself having been uneventful. The onset of the milk fever was usually abrupt; the first symptom was excitement, and this was followed by tetany, which might be confined to the hind limbs, or might involve all four limbs. Convulsive seizures, when they occurred, rapidly led to coma. In 1897 Schmidt discovered that the injection of fluid into the mamma produced prompt and complete cure. This injection method was later replaced by inflation of the breast. These measures had reduced mortality from between 60 per cent. and 70 per cent. to less than 1 per cent.—a remarkable result. It was not clear how mammary inflation produced the cure. In 1924 jointly with Prof. H. Dryerre, Prof. Greig had formulated a working hypothesis in which it was suggested that the essential condition was a parathyroid deficiency with a fall in the blood calcium, the latter deficiency being accentuated by lactation. The colostrum of the cow revealed a very high concentration of calcium. Mammary inflation produced, he submitted, a stimulation of adrenal secretion, and consequently increased the excretion of any toxins which might be present;

or it caused a reduction of the milk secretion and a further transference of calcium from the blood to the milk. The curative effect of inflammation of the mamma was due to the restoration to that organ of its due blood calcium. Once an animal had been attacked by this malady there were likely to be recurrences at the subsequent calvings. In every case of milk fever investigated the blood calcium was deficient. Moreover the progress of cure, as manifested in the disappearance of symptoms, corresponded with the rise in the blood calcium, which remained abnormally high for five days. As would be expected, the injection of calcium salts cured the condition. The recovery of the cow from its torpor and helplessness after treatment was dramatic; very definite improvement was noted in five minutes, the change being especially rapid when the injection was intravenous.

The calcium deficiency might have its basis in a parathyroid deficiency, or there might be a great loss of calcium in the milk. There was as much calcium in half a gallon of colostrum as in the whole of the soft tissues of the body. Possibly mammary distension cured the disease by preventing a further transference of calcium from the blood to the milk. In ewes there occurred a "lambing sickness" which was much akin to the condition just mentioned. Investigation of a series of cases showed the following blood calcium contents:—

mg. per 100 c.cm. of serum.	Normals.	Lambing sickness.
Maximum ..	11·92	5·94
Minimum ..	7·92	3·51
Average ..	9·80	4·40

These figures were confirmed in an investigation by Sjollema. Mares suffered from a similar disease, and they responded with equal suddenness to mammary inflation or injection of calcium salts; the same was true of the parturient goat, sow, and bitch.

As milk fever appeared to represent a metabolic upset and seemed to occur in all domesticated animals, it was reasonable to assume that a similar condition occurred in woman, and there were parallel symptoms in eclampsia. The blood condition in the two was, however, markedly dissimilar. In serious cases of eclampsia inflation of the breasts gave no response, and the results of calcium injections in this disease had not been encouraging. Yet a case of eclampsia recorded in 1931 by Dr. J. G. Kinnimonth suggested that a condition which was pathologically related to milk fever in domestic animals did occur in the human; the symptoms here closely resembled those of milk fever in the cow, and prompt and complete recovery followed inflation of the breasts. There was also some parallelism between eclampsia and a disease in ewes known as pregnancy toxæmia.

Prof. DRYERRE, who had been investigating ewes, said that the ewe was sometimes affected before the lamb was born. He was convinced that many cases of pregnancy toxæmia in ewes were confused with those of lambing sickness. Some writers had had no success with calcium injections in lambing sickness, but Prof. Dryerre suggested that their cases were really those of pregnancy toxæmia. Some confusion was natural, since in both conditions the animal was unsteady of gait, and liable to fall, and might be almost comatose. Calcium salts had no effect in the toxæmia of pregnancy—indeed, their injection might hasten the fatal termination. In an investigated series there was no significant difference in the blood calcium or the potassium content; between the normal animal and the animal suffering from pregnancy toxæmia; the magnesium showed a slight difference, and the inorganic phosphorus in pregnancy toxæmia was lower than the normal. There was a fall of blood calcium in eclamptic cases, but not to a significant extent, and it was thus not likely to be a causal factor.

Prof. E. C. DODDS referred to an investigation he had conducted into tetany as manifested in some Welsh mountain ponies, which had been captured on the hills and transferred in railway trucks. These animals developed an illness bearing a clinical resemblance to milk fever; their blood calcium was low, and restoration followed the giving of calcium intravenously or subcutaneously. In these animals there was no calcium loss to account for the low blood calcium; his view was that the fright induced by their capture and the railway journey induced an alkalosis. If so, there was no need to postulate a loss of calcium in the milk. He considered that any attempt to establish a parallelism between the milk fever of cows and eclampsia in the human was doomed to failure. While in milk fever the blood calcium was said to be always low, in eclampsia the blood calcium might or might not be low, bile pigment might be in the blood, or the blood-urea might be high. The other great difference between milk fever and eclampsia was in the blood pressure. The latter provided the best indication to the prognosis in a case of eclampsia. Apparently in the cow the blood pressure was not high after inflation of the udder. From a search of the literature, he had come to the conclusion that the only condition comparable in women to milk disease in cows was lactation tetany, which was sometimes found in women who prolonged lactation for nine months, or over. He asked whether alkalosis was considered a reasonable explanation of the symptoms in domestic animals; it seemed to be easily excited.

Mr. W. L. LITTLE said he had had experience of milk fever in the cow, the goat, and the dog, and was able to trace a number of disabilities following upon insufficient diets. The cow needed much lime. He felt sure that an excess of phosphates interfered with the assimilation of the calcium, and in this country the diet of most cows was fairly rich in phosphates. As milk fever was prevalent on some farms and very rare on others, he suggested that the excess phosphorus taken might interfere with the calcium assimilation. The best results had followed winter feeding on a combination of cod-liver oil and calcium salts. Injection of calcium salts just after calving prevented the onset of the disease on farms where it was prevalent. Autopsies on a number of cases of milk fever showed that in most of the cases the death was from septic pneumonia. In deaths occurring after very severe attacks there were changes in the muscles, the affected muscles showing a dry necrosis, and blood extravasation between the fibres. In the kidneys and bladder small hæmorrhages were evident. He advised that calcium injections should be given very slowly.

Mr. O. STINSON spoke of his experiences with cattle on two farms in Westmorland very dissimilar in the matter of soil. In one the cattle showed a distinct aphosphorosis; if the animals were transferred to the other farm for a time these symptoms disappeared.

Prof. F. J. BROWNE did not feel able to accept Prof. Greig's explanation of interpretation. Though there might be hypoglycæmia in the blood of these animals, he did not think the essential pathology of the disease was due to that. What was the cause of the deficiency in the function of the parathyroid gland? He thought the condition in cows, though not

identical, was closely allied to the eclampsia found in women. In women the toxæmias of pregnancy occurred chiefly in strong and well-developed multiparæ; in cows also the disease occurred mostly in strong good milkers, not in the first pregnancies, but after two or three calves had been born. In cows which yielded 30 to 35 litres of milk a day the whole blood content of the animal was no more than 45 litres, and it had been suggested that in these cows the blood became too concentrated. When thinking of the enormous quantity of milk excreted, it was easy to picture how much blood must pass from the liver into the systemic blood; in eclampsia, he believed, there was too great a rush of blood through the capillary paths of the liver, causing a poisoning of the general system. Prof. Greig had said he did not find a rise in the blood pressure after carrying out inflation of the mamma. But if one occluded a large capillary area, a rise of blood pressure must ensue, and if this was not evident, it must mean that there was an increased flow through some other peripheral part. After mammary inflation there was greater excretion and a diminished flow through the portal system; the systemic blood became purified, so that the animal got well. As to the way in which calcium acted, it had been shown that carbon tetrachloride poisoning in dogs could be prevented by a prior administration of calcium; Prof. Browne thought that the only way the calcium could act was by stimulating the hepatic cells. Lack of calcium resulted in paralysis of tissue cells, so depleting the efficiency of the liver cells which were stimulated by calcium therapy.

Prof. F. W. WOOLDRIDGE said he had often seen a sudden restoration of consciousness in cows as a result of inflation of the udder in milk fever; the difference produced in twenty minutes was very impressive. If the condition was one of hypoglycæmia, it was difficult to understand how inflation of the udder could influence it. In his view hypoglycæmia was one of the effects of the disease known as milk fever, not the cause. If that was so, Prof. Browne's theory as to the action of calcium on the liver cells fell to the ground as an explanation of the sudden recoveries after inflation. He did not think the cause of the condition had yet been found. Recently he saw a milch goat which had been a very heavy milker. Five months previously a kid had been born to it. It was fatigued after a long railway journey, refused its food, and the milk fell off almost to nil. After three days it showed all the symptoms regarded as typical of milk fever. He decided to do udder inflation empirically; within fifteen minutes the goat, from being apparently dead, raised its head, took cabbage, and a few minutes later was standing on her feet. His own suggestion was that the disease was a neurosis of some kind, though he had no theory as to how it was produced.

SECTION OF ORTHOPÆDICS.

AT a meeting of this section, held on March 1st, the chair was taken by the President, Mr. HARRY PLATT, and a discussion on

Fractures Round the Ankle-joint

was opened by Mr. R. BROOMHEAD. He classified fracture dislocations of the ankle-joint into adduction and abduction fractures. Both were, he said, due to indirect violence, the former to torsion and the latter to simple leverage. There were three degrees of the abduction type: (1) when the internal lateral ligament was intact; (2) when the ligament was torn or the malleolus fractured; and (3) when, in addition, the astragalus was misplaced or the posterior margin of the tibia was fractured. The third degree type proved difficult to reduce, and restoration of function was hard. Treatment depended on the displacement. Those without displacement did not require immobilisation. Walking might be commenced early, with a firm though mobile support. When there was displacement, reduction was easy if it was possible at all; the others usually required open operation. Inversion should be obtained by inverting the back-foot and not by twisting the fore-foot. Adduction fractures were much more unstable, and should be fixed at a right angle. One of the best-known and most widely used methods of ambulatory treatment was Delbet's. Patients could walk about quite well if a special shoe were fitted over the plaster. Other forms of treatment included Böhler's, which was one of the most practical, although he did not allow walking until all swelling had subsided. Plaster was ideal as a protective splint, but not so useful as a corrective, and fractures with displacement were better treated in bed. There was no evidence that return to work was possible any earlier after the plaster methods.

Operation was to be advised in some third degree abduction fractures, some second degree adduction fractures, and all compound fractures. The skin of the foot had great potentialities for sepsis, and operation should be delayed until the skin had been prepared and the swelling had subsided. In adduction fractures the internal malleolus and in abduction fractures the external malleolus must be fixed, and often the posterior tibial margin as well. One alternative was to fuse the ankle-joint after all swelling had subsided—in about a fortnight; the other was to do all that could be done by conservative treatment at first, and then to fuse the joint in six months' time if function was inadequate. Vaseline pack dressings had proved very satisfactory for compound fractures. Amputation was often worth consideration as an immediate measure, as it saved a long and painful period of treatment with a useless joint as the result. It was very important—and often difficult—to get early and complete reduction in cases of separated epiphysis.

General anæsthesia was preferable; local anæsthetics had proved disappointing. Some 20 c.cm. of fluid had to be introduced into an already swollen limb, and often spasm of the peroneal muscles was not abolished by the injection, so that the displacement recurred as soon as the hands were removed. These fractures had great importance in relation to compensation claims. Return to work should be possible in eight to ten weeks. If there was displacement with marginal fracture the patient was usually disabled for 16 to 18 weeks, and often up to six months.

FRACTURES OF THE ASTRAGALUS.

Mr. J. P. HOSFORD said that he had only found 15 cases (in 13 patients) of fracture of the astragalus at St. Bartholomew's Hospital in the last ten years. The neck was the part most commonly broken, and the ankle-joint might or might not be involved. The cause was seldom a direct blow as the astragalus was so well protected, and in twisting strains the malleoli fractured more easily than the astragalus. The most usual history (in nine cases) was a fall on to the heel from a height, and often there was some other gross lesion in the tarsus or a subastragaloid dislocation. There were four types: fractures of the neck; fractures of the posterior process; crush-

fractures of the body, and chip fractures. Pain might be caused by bony block due to displaced fragments. In a severe case the symptoms resembled those of a Pott's fracture; the hollows on either side of the tendo Achillis might be filled if there had been backward displacement. Some cases could only be diagnosed radiographically. The treatment was immobilisation in a plaster cast and rest for eight to ten weeks. If the head was displaced medially it must be manipulated into position before the plaster was applied. Reduction of a posteriorly-displaced fragment might need open operation. The function in unreduced cases was very bad. Fractures of the body were generally irregularly comminuted, and each case must be judged on its merits. If there was bony block the fragment must be removed. Any sub-astragaloid dislocation must be reduced. Amputation was necessary in open fractures where the astragalus was pulped. Operation was required in about half the cases, and there was in most cases some permanent disability of the ankle.

SEPARATED TIBIAL EPIPHYSIS AND CHRONIC ARTHRITIS.

Mr. E. P. BROCKMAN described a new method of treating those cases in which growth of the tibia had ceased after separation of the epiphysis but growth of the fibula had continued, distorting the foot. He did an osteotomy of the tibia, plugging it with the growing end of the fibula, which he removed. Since growth in the tibia had ceased, there seemed no harm in stopping growth of the fibula, and the results had been satisfactory.

If a case of old Pott's fracture developed constant pain in the ankle, operation was indicated, and there was no reason for delay. Mr. Brockman advocated arthrodesis, and described his method for these cases, mentioning good results in 12 cases.

DISCUSSION.

The PRESIDENT described a survey he had made of ankle-joint fractures at the Ancoats Hospital in the decade 1920–30. The treatment had responded to the dictates of surgical fashion. In the first four years the handling had been conservative: manipulation, plaster fixation, and very late weight-bearing. The ultimate industrial results had been satisfactory. For the next three years the Delbet plaster had been used both as an ambulatory splint and as the primary method of fixation, early weight-bearing being encouraged. With earlier weight-bearing there had undoubtedly been earlier return to work. During the last two or three years the Delbet plaster had been given up on the grounds of expense, and Böhler's skin-tight complete plaster had been used, and the patient allowed to walk early, either on a stirrup or on a slab of plaster. The Delbet method could be very successful as a method of primary fixation. Prompt reduction could be followed by perfect restoration, and there was little tendency to redisplacement. Mr. Platt saw no place for primary open operation.

Mr. G. PERKINS preferred Ashurst's classification of these fractures in ætiological groups: (1) External rotation, causing fracture of the internal malleolus and a spiral fracture of the lower end of the fibula, often with backward dislocation of the foot. This was easy to reduce and no disability followed. (2) Abduction, due to an abducting force on the foot, causing a high transverse fracture of the fibula, and breaking off the internal malleolus—rather difficult to reduce and very apt to recur. (3) Adduction—not very common, difficult to reduce, and often needing operation. (4) T-shaped into the joint, due to a vertical force passing up the leg. Each type required different treatment and offered a different prognosis.

Mr. N. L. CAPENER mentioned a late result of fracture of the astragalus: compression changes, presumably due to interference with the blood-supply, followed by sclerosis and compression.

Mr. DUDLEY BUXTON thought that fracture of the lower end of the fibula with no displacement whatever was by far the commonest type. It required support for six or eight weeks. He suggested that ambulatory treatment had become popular at a time when every hospital was experiencing pressure on its beds. Immediate reduction was of the first importance, followed by fixation in an apparatus to keep the fracture reduced. The advantage of the stirrup, or a good thick plaster support, was that it enabled patients to walk without crutches. He kept plaster on for eight weeks in cases which had been perfectly reduced, and a week or two longer if there had been a slight error in reduction. Some feet would swell however they were treated; and Elasto-plast bandage was useful if swelling persisted.

Mr. ROBERTS advocated the Delbet plaster for its simplicity, its allowance of early dorsiflexion and early ambulatory treatment without crutches, its comfort, its self-massaging action, and the absence of swelling. It must, he said, fit the patient very accurately, being moulded to the whole limb, including the malleoli and the external peroneal nerve. There was less tendency to osteo-arthritis if the patient got about early. A fracture was in a good position if the astragalo-tibial line was horizontal. The use of a needle for local anæsthesia made a fracture compound.

Mr. NAUGHTON DUNN said that reduction and prolonged fixation were the secret of the treatment of all fractures. He had never plated any fracture. Most speakers had advocated too brief a period of fixation. The result depended partly on the type of patient; he agreed that arthrodesis offered the best course if pain persisted.

Mr. ROWLEY BRISTOW supported Ashurst's classification, and agreed that for the first type complete and early reduction was absolutely essential. So long as the fracture was reduced, the subsequent treatment did not matter. Anæsthesia should always be general. In the second type the difficulty of reduction was nothing to the difficulty of keeping the fracture reduced. This type required prolonged fixation, followed by raising of the heels, an outside iron, and a T-strap. The difference between prognosis and treatment in the two types illustrated the value of the classification.

Mr. BROOMHEAD replied.

CHILDREN'S WARD AT BECKENHAM HOSPITAL.—Sir George Sutton last week opened the children's ward which, at a cost of £6000, he has presented to Beckenham Hospital. Instead of four cots in the adult wards there are now 12 cots and beds. The addition has been named the "Ruth Sutton Ward" in memory of the late Lady Sutton.

"LAW TOUTS" IN HOSPITALS.—The attention of hospital managers has been called by the Incorporated Law Society to the frequency with which "touts" for certain solicitors visit accident patients in hospitals and endeavour to persuade them to sue for damages. The practice is to suggest that if the case is placed in the hands of the "tout" he will do the work free of charge in the event of failure, and will charge only two shillings in the pound when damages are obtained. Should, however, the damages be substantial the solicitor's remuneration is likely to be much larger than the costs he could properly charge.

REVIEWS AND NOTICES OF BOOKS

Diphtheria, Past and Present.

Its Ætiology, Distribution, Transmission, and Prevention. By J. GRAHAM FORBES, M.D., F.R.C.P., D.P.H., Principal Assistant Medical Officer, London County Council. With an introductory note by Sir FREDERICK ANDREWES, M.D., F.R.S. London: John Bale, Sons and Danielsson, Ltd. 1932. Pp. 832. 45*s*.

Dr. Graham Forbes presents in this volume of some 800 pages what is probably the most comprehensive account of diphtheria, considered from the public health and preventive aspects, which has ever been attempted. As regards prevention, at least, it is almost literally accurate to say that the survey extends from China to Peru. The last publication on diphtheria comparable in importance with the present work was the monograph by several authors, issued by the Medical Research Council in 1923. In that monograph diphtheria was discussed chiefly from the bacteriological and pathological aspects, although the methods of specific prevention then available were brought under review; the last few years have seen diphtheria added definitely to the list of preventable diseases. Dr. Graham Forbes divides his book, which is founded upon his Milroy lectures of 1929, into four sections dealing respectively with ætiology, distribution, transmission, and prevention. Each section is abundantly furnished with maps, charts, and statistical tables which add to the value and clearness of the text. The section on prevention occupies about half the book, and rightly so, since it is by far the most important.

The first three sections present a very complete picture of the natural history of diphtheria in England and Wales. The incidence of the disease in the counties and large cities is discussed in detail and a wealth of epidemiological data is supplied. These sections form a background, which, it is to be hoped, may come to be chiefly a historical background to the section on prevention. Here the first steps in the displacement of diphtheria as one of the endemic lethal diseases of childhood are described, and the measure of achievement is recorded, country by country, county by county, and town by town.

It is satisfactory to learn the extent to which active immunisation has already been adopted in this country. As one of the most tireless apostles of the specific prevention of diphtheria, Dr. Graham Forbes must have been gratified to observe the increase in the numbers of children protected since the publication of his Special Report to the Medical Research Council No. 115 in 1927. Active immunisation has now at least been commenced in a large number of towns, great and small, in England and Wales. Scotland, and particularly Edinburgh, early realised the advantages of a child population immune to diphtheria. France and America are well ahead of us in this matter; in those countries active immunisation against diphtheria has been adopted with something more than tolerance and in some towns in the United States even with enthusiasm. Though Schick's test for immunity is acknowledged by all competent observers to be valid, its performance is by no means essential as a preliminary step. The omission of the initial test merely results in the "immunisation" of a few children already immune, and since the measures adopted are simple and can do no harm, this does not matter. Indeed, since with the extension of the practice of immunisation some of the work must inevitably be carried out by practitioners with little experience of the test, the omission may be wise, and it has the further advantage of reducing the number of attendances of the mother and child. Active immunisation by the injection of one of the preparations described by Dr. Graham Forbes may be started at the first visit. The second injection follows, preferably, three weeks later, and the third and last a fortnight after that. A confirmatory Schick test is desirable when possible after attempted immunisation.

The preparations now used in this country are said to be devoid of risk. Reactions, whether local or general, are rare, and there is no negative phase. Toxoid (Glenny), the anatoxine of Ramon, is diphtheria toxin which has been digested with formaldehyde. The resulting product, whilst completely non-toxic, retains unimpaired its power of stimulating the production of antitoxin. Toxoid, with or without antitoxin, in soluble or in floccular form, has completely replaced, in Great Britain and France at least, the old toxin-antitoxin mixtures. Dr. Graham Forbes supplies ample evidence of the results of active immunisation against diphtheria. Whether it is applied to specially exposed adults, such as nurses in fever hospitals, or to children in orphanages or homes, school-children or children below school age, the reports are the same. Whenever the measure has been introduced upon a sufficient scale there has been a notable diminution in the incidence of diphtheria.

The task which Dr. Graham Forbes set himself was onerous. He has performed it with conspicuous ability and faithfulness and his work is an important addition to the literature of preventive medicine. The labour involved in the collection and analysis of the facts compels admiration. No public health department should fail to add this volume to its books of reference, and it is to be hoped that members of public health committees, if they do not purchase, will at least borrow it.

A Manual of Surgery.

By ALEXANDER MILES, M.D., LL.D., F.R.C.S. Edin., Consulting Surgeon, Royal Infirmary, Edinburgh; and D. P. D. WILKIE, F.R.C.S. Edin. and Eng., Professor of Surgery, University of Edinburgh. Vols. II. and III. London: Humphrey Milford, Oxford University Press. 1932. Pp. 685 and 578. 12*s*. 6*d*. each volume.

THESE two volumes complete the new Thomson and Miles surgery, now under the combined authorship of Mr. Miles and Prof. Wilkie. We have little to add to our review of the first volume. Seen as a whole, it is a very satisfactory work from many points of view. The authors are adepts at providing just the amount and kind of knowledge the average undergraduate wants. They have shown judgment both in the selections and rejections, and the presentation of material is singularly free from bias. The volumes are of convenient size, not too heavy to hold in the hand, and the print is easy to read, whilst the numerous illustrations must materially assist the student's understanding of surgery. A pass examination should have no terrors for a candidate who knows his Thomson and Miles, and he will have

gone very far towards reaching the standard required in the higher surgical diplomas and degrees. One small criticism suggests itself. Whilst the illustrations serve their purpose of demonstration fairly consistently, they nearly all seem to be half-tone blocks reproduced from photographs and are not nearly so attractive as the drawings which were common in the older editions. Moreover, some of the old illustrations seem to have been photographed and to have rather lost value in the reproduction. In all other respects the authors deserve congratulations on having completed their task of revision in an eminently satisfactory manner.

Die Dickdarmschleimhaut, Ihre Normale und Pathologische Funktion im Roentgenbilde.

By Priv.-Doz. Dr. WERNER KNOTHE, Head of the Radiological Department of the II. Medizinischen Universitäts-Klinik der Charité, Berlin. Leipzig: Georg Thieme. 1932. Pp. 56. M.8.

THIS small volume on the changes in the mucous membrane picture of the large intestine follows closely on the work recently published by Albrecht on the mucous membrane of the stomach. Both are inspired by Hans Berg, who followed up the pioneer work of Forssell and brought it into every day use. Dr. Knothe has recorded here his observations on the mucous membrane of the large intestine. His study is beautifully illustrated by many radiograms of a high standard, which show clearly the different forms and varieties of mucous membrane as seen in relief picture. It is a good piece of work, but the author does not always convince us (except in regard to certain well-recognised conditions) that the appearances shown are necessarily produced by the pathological or disordered physiology to which they are ascribed—e.g., we do not know that it is "irritation" that produces certain changes in the relief picture. It is a moot point whether a pathologist would recognise "irritation" of the mucous membrane except when it is severe enough to cause some degree of inflammation. Opportunities for control are rare. The proof of cause and effect sometimes appears to be slender, especially when we consider the extraordinary variations of the normal that must be possible in such a mobile structure as the living mucous membrane. It seems possible that Dr. Knothe may have gone too far in the interpretation of his shadow pictures. In spite of the obvious difficulties, work of this kind should be pursued for a long time on subjects who may be presumed to be normal. It is only by the study of normal physiology that relatively slight changes in the relief picture of the mucous membrane can be interpreted correctly and deductions on disordered physiology made.

The get up and standard of reproduction of this book are very satisfactory, as in fact are all the radiological books that we have seen coming from this publishing house.

Abnormal Psychology.

By H. L. HOLLINGWORTH, Ph.D., Professor of Psychology, Barnard College, Columbia University. London: Methuen and Co., Ltd. 1931. Pp. 556. 15*s*.

Prof. Hollingworth has attempted, with a considerable measure of success, to cover the whole field of psychopathology from the study of feeblemindedness to drug addiction. She deals historically with the various theories of neurosis, and such neglected writers as Janet, Babinski, Hurst, Rosanoff, and Rivers are given due attention in addition to the more familiar Freud, Adler, and Jung. The volume is comprehensive and there are very few conditions and psychological theories which are not described and criticised. The most critical pages of all are devoted to the Freudian psychology; the fact that references to this school are directed against the "psycho-analogical viewpoint" will irritate many readers. It is obvious that the investigation of psycho-analysis which underlies the criticisms has been extremely superficial, with the result that Freud's concepts of unconscious symbolism and the use of dream analysis are regarded as mysticism and demonology. The very fact, as Prof. Hollingworth states, that psycho-neurotic disorders have been cured by all forms of charlatanry and suggestion may invalidate every theory of explanation, but she appears to forget that psycho-analysis itself has not failed to give a reasonable explanation of the mechanism of suggestion and the charlatan. The simple theories which she prefers to psycho-analysis are simple largely because she has made them so, and has not subjected individual cases to prolonged investigation. The findings of psycho-analysis satisfy not only the problems of psychopathology to a high degree, but shed a light upon many human institutions. On this latter support of psycho-analysis she makes little comment. No theory has finality, but up to date the Freudian hypotheses are based upon as prolonged and as scientific an investigation of the facts as any other theory, and seem to explain more of the facts.

Emigration, Migration, and Nomadism.

By the late WALTER HEAPE. Edited with a preface by F. H. A. MARSHALL. Cambridge: W. Heffer and Sons. 1931. Pp. 369. 12*s*. 6*d*.

A PLEASANT portrait of Heape forms the frontispiece of this characteristic book. He was originally an embryologist; students of Quain's Anatomy will remember his pictures of the early development of the mole. But before he was 35 he had broken away from the conventional methods and became a pioneer in the study of the physiology of generation, activating en route Dr. F. H. A. Marshall, who edits this book, which Heape left a little short of completion when he died in 1929. He deals with the journeys of animals from which a return may be anticipated (migrations), their movements by which they permanently evacuate a territory (emigration) and their wanderings at random, and incidentally an extraordinary range of germane and allied topics in which any naturalist or student of human behaviour will be much interested; man himself is excluded from the scheme. The background on which all this wealth of detail is arranged is sexual physiology and Heape's inductive belief in some kind of generative ferment which animals could apparently absorb from their surroundings; we can imagine the delight with which he must have welcomed Evans and Burr's discovery of vitamin E in 1925. We call the book characteristic of Heape because it is gentle, humane, and scholarly. It is also exceptionally interesting, whether it deals with the bear's conception of territory, or with the migrations of Painted Ladies, or tells how Stefansson sat over his seal hole in perfect confidence that his daily bread must sooner or later come up to take a breath of air.

THE LANCET.

LONDON: SATURDAY, MARCH 5, 1932.

YOUNG OFFENDERS.

In 1927 the Government of the time appointed a Departmental Committee on the Treatment of Young Offenders. It was then recognised that the reformation of the offender had become the keynote of the administration of justice, and that proportionately greater stress should be placed upon the need for reformation of the young offender during the years of mental plasticity. As the result of their careful deliberations, the Committee came to certain definite conclusions of which the following were some of the most important. First, that the magistrates sitting in the juvenile courts should be specially qualified for the work, that there should be a pooling of their experiences, and that they should make themselves fully acquainted with the institutions to which children are sent. Secondly, that the whole procedure of such courts should be remodelled in the direction of simplicity; that the court should be in possession of full medical and scholastic reports, should work in close coöperation with educational authorities, and should not be situated in premises used for other courts, and that these latter should be able to refer a person under 17 to a juvenile court. Thirdly, that much better facilities should obtain for the examination of young offenders, and that at least three central remand homes should be established for this purpose, but wherever possible the fullest use should be made of bail. The Committee also stressed the importance of probation in the open rather than in institutions; the wider use of hostels was urged. No sentimental ideas however were encouraged with regard to probation; supervision by women for girls, and men for boys was recommended. The Borstal system was praised, but it was urged that children should be sent there for training and not "sentenced to detention under penal discipline, &c." The age for capital punishment was recommended to be raised to 18. As regards mental defectives, the grave lack of accommodation was emphasised, and the pathetic case of the post-encephalitic offender was mentioned as calling for special treatment on purely medical lines. While these points do not exhaust all the excellent recommendations made by the Departmental Committee they do include all the points of interest to medical men who are or may be directly concerned with such children as come before juvenile courts.

The Children and Young Persons Bill now before the House of Commons is substantially based upon the recommendations of the Committee of 1927.

In future the court shall not be associated with the premises of a police station or adult court, and the magistrates are to be chosen from a panel of those specially qualified to deal with juvenile cases.

The age of young offenders or those needing specially guardianship who appear before a juvenile court will be raised to 17.

In the place of the two categories of schools (industrial and reformatory) there shall in future be one class (approved schools), to which children are to be sent for a period of three years, but if under 11 years of age they may be detained until the school-leaving years.

The age of capital punishment is to be raised to 18, and the adult courts are to have the power to remit juvenile offenders to the juvenile court.

But while the Bill illustrates the fact that time has worked a change towards the appreciation of the special needs of young offenders, it has singularly failed to take into account the medical, psychological, and sociological advances that have been made in the field of juvenile delinquency. Pædiatricians, psychiatrists, and social workers who are in daily contact with the neurotic and behaviour disorders of children and of young adolescents, are witness to the multiple factors that go to make up the mentality of the so-called difficult child. They are aware that while society must expect of its members a reasonably high degree of conformity to social usages, it is impossible to apply to young people the same legal categories as are applied to adults. Moreover, during the years of mental plasticity, before social sentiments are established, the child as a labile organism is only too easily influenced by medical conditions, sometimes gross, sometimes subtle, and by environmental factors. Above all, there are individual reactions to family stresses born of temperamental peculiarities which have to be carefully assessed before remedial measures can be applied. Delinquency as much as neurosis is a function of the personality in its interplay with environmental forces. Only expert study of this interplay can reach an understanding of the forces at work and apply a suitable remedy. In Belgium, the Government has established at Moll an institution which is nothing less than a research station; to this young offenders are sent and here their problems are studied by an expert staff before they are sent to places of detention according to the character of the disorder from which the child and adolescent is presumed to be suffering. There is unhappily no indication in the present Bill that any consideration has been given to the all-important subject of diagnosis upon which adequate treatment shall be based. In some of our juvenile courts, the presiding magistrates have invited the coöperation of psychiatrists who have experience in child psychology from their practice in the child guidance clinics and analogous treatment centres. Their value has been proved in many cases. They have sometimes in a side room of the court—or more often in their clinics when cases have been put on bail or probation—disclosed medical and psychological factors which explain the offence. They have discovered family conflicts and environmental stresses from which the child in his dilemma has escaped along the path of delinquency. In addition they have been able to give valuable guidance to probation officers

in charge of cases, and have saved curable cases from years of confinement in reformatories and industrial schools where no classification can be made, or rarely is made, and where the treatment must be more or less standardised.

The outstanding omissions from the Bill are therefore, first, the need for observation centres in the place of the present remand homes, at which centres diagnosis of the condition can be made after expert social and psychological data have been collected. Secondly, the need of the presence in an official capacity in juvenile courts of trained psychiatrists to aid the magistrate in disposing of cases after the charge has been substantiated. Lastly, no provision is made in the Bill for the establishment of an adequate service of probation officers trained in the ground-work of family psychology and social conditions and possessing the type of personality which is only too rare in this type of work. Many who do this work in connexion with various voluntary bodies are poorly paid and succeed largely because of their enthusiasm for work of deep social importance. Those in authority must surely recognise that the progress of modern pædiatrics and psychology is not the work of yesterday, but is the result of the accumulated knowledge of the last 50 years. The philanthropic zeal of the prison reformers has been richly supplemented by a growing body of knowledge with regard to the motives of disorders of conduct. The foundations of personality are laid in childhood, and to correct the disorders of personality in the early years of childhood or at least in adolescence is to lay the foundations of good citizenship and so to relieve the courts of adult cases who return again and again because early measures have not been adopted to deal with social evils at the root.

THE DIAGNOSIS AND TREATMENT OF POLIOMYELITIS.

POLIOMYELITIS does not present quite the same problem in England as it does in Australia, but no one can afford to ignore the lessons of the Melbourne epidemic. Dr. JEAN MACNAMARA and Dr. F. G. MORGAN conclude their account of it in our present issue, and their paper defines very clearly our responsibilities towards victims of this disease. The story is highly encouraging, but Melbourne has evidently set a standard to which we cannot attain without a definite effort. In Australia the reward of such effort has been very great.

The practitioner will find the report interesting, first because it brings out clearly the possibility of diagnosis before the stage of paralysis and describes in detail the means by which this early diagnosis can be made. Attention is directed towards the signs and symptoms of the prodromal feverish attack and especially to the "spine sign" which signifies impending paralysis. Lumbar puncture, it is emphasised, must be undertaken only if the warning indications of cerebro-spinal involvement are present. Premature disturbance of a sick and feverish child is well avoided, but it is not discomfort only that is to be feared. For lumbar puncture, done in a genuine case of poliomyelitis before the meningeal irritation has provoked the characteristic reaction, will be misleading and may also upset the delicate relations between the cerebro-spinal fluid and the pia-arachnoid and thus favour spinal infection. That lumbar puncture tends to increase the permeability of this last line of defence is insufficiently appreciated; indeed, early puncture is often ardently advised. Evidently this is to be deprecated; on the other hand it cannot be too strongly emphasised that lumbar puncture in a suspected case of poliomyelitis is urgently required as soon as there are signs of the central nervous system being involved. Dr. MACNAMARA is also emphatic that the examination of the fluid should be done there and then, at the bedside, and that immune convalescent serum should be injected without removal of the needle if the diagnosis is positive. Delay in giving serum may be very serious or disastrous. But if the supply of serum is unavoidably delayed it seems to be better to await its arrival, and then do the lumbar puncture, rather than prove the diagnosis by lumbar puncture and then wait for the serum. The serum itself must be free from any toxic or irritating substance, so that neither a general febrile reaction, nor a local spinal or meningeal irritation, may disturb the clinical picture. The result of an adequate dose should be a prompt and progressive fall of temperature and clinical improvement; unless this takes place a further dose must be given promptly, and the decision is not at all easy to make if the serum has properties beyond its immunising effect. MACNAMARA and MORGAN therefore prefer human to horse serum, and they use serum prepared and preserved with the utmost precaution but without antiseptics. Obtained from old cases, it is pooled and tested from time to time against virus. Its therapeutic powers are almost magical. On the system which Dr. MACNAMARA adopts, the aggregate paralysis of each case can be expressed in figures and the contrast between treated and untreated cases is astonishing. The records are all the more convincing in that the majority of serum cases show a paralysis aggregate numeral, though a very small one, indicating that the virus succeeded in reaching the anterior horn cells and that they were true, not abortive, cases of poliomyelitis.

England seems to have been the first European country to harbour the disease (1784, UNDERWOOD) which was said to be of Asiatic origin. It was not recognised until 1840 on the continent (HEINE) and until 1841 in America (MEDIN). We had indeed a start, but probably a short one, and this is of interest because some hold that we have become relatively immune as a race and therefore need not fear the serious epidemics from which other countries suffer. Whether the prolonged endemic persistence of poliomyelitis has given some degree of immunity, or the cool climate is in our favour, we do not know, but it would be easy to have an undue sense of security. There have been various small epidemics in this country in the last five

years and their scale might easily become larger. The cost in death and disability has not yet been very great, but it should be generally realised that a high proportion of our endemic cases are of the epidemic (acute febrile) character, and therefore susceptible of preparalytic diagnosis and treatment. The means should be ready to hand

USE OF TUBERCULIN IN DIAGNOSIS.

NEARLY forty-two years have passed since KOCH introduced the substance now universally known as "old tuberculin." It failed to live up to expectations as a specific cure for tuberculosis, and seven years later KOCH himself abandoned it in favour of "new tuberculin." From that time onwards the ingenuity of the immunologist has steadily added to the number of tuberculins until confusion has become worse confounded. But whatever may be the verdict of posterity on their value as remedies, it seems pretty clear that KOCH'S old tuberculin is useful in diagnosis. Of the tuberculin tests used in clinical practice the one introduced by PIRQUET in 1907 was *facile princeps* until after the war. It has passed through many vicissitudes of obloquy and praise and, although leading exponents of tuberculin diagnosis, such as HAMBURGER, have demonstrated its fallacies, it is still extensively used in many lands, though not very largely in this country. In 1908, MENDEL and MANTOUX independently introduced the intradermal test, which, though admittedly more precise, failed to attract much attention until after the war. Since then it has come to its own as the most reliable and accurate of the tuberculin tests, largely as a result of extensive records compiled in America. In a valuable report issued last week, Dr. P. D'ARCY HART [1] has explored much of the new work and condensed it—a very considerable achievement—and also records a tuberculin survey of London children which may be compared with those recently published by W. F. GAISFORD from the East London Hospital for Children and by D. J. DOW and W. E. LLOYD from the Brompton Chest Hospital. A glance even at Dr. HART'S bibliography leaves a feeling almost of dismay in the minds of those familiar with the solid, red-paper-covered volume of BANDELIER and ROEPKE which was the great stand-by of the early days.

With some of the issues raised by these modern developments we have already dealt.[2] Three at least may be underlined as important: (1) the relative value of the various fractions of crude tuberculin which have been isolated during the last few years; (2) the records of the tuberculin surveys as evidence of the prevalence of tuberculosis in both large crowded communities and in small isolated units; and (3) the sociological significance, from the standpoint of the immunised community, of the negative reaction, especially as regards the "contacts." To these, Dr. HART would presumably add a fourth, for he asks the reader to reconsider the tuberculin skin reaction as a means of clinical diagnosis in relation to the new Mantoux test. The first question is of a highly technical character, and must be left to the immunologists and biochemists to work out, but light is thrown upon it by Dr. HART'S observations [3] on the incidence of skin sensitiveness to glycerinated veal peptone broth (the medium used in preparing old tuberculin) and its relation to tuberculin sensitiveness. Of the second question more needs to be said. In surveys of whole communities, especially of school-children, the intradermal test has been used to amass vast columns of statistics. It was necessary that it should be done for each country, but we confess to the hope that the spate of uninspired arithmetic will soon diminish, for we doubt if a few odd thousand more children added to the tally will help matters much. Probably most clinicians of experience will agree with the view that a positive tuberculin reaction means that the person has at some time been affected with the tubercle bacillus, but, as Dr. HART admits, no more stringent conclusions can be drawn than this. Comparing some of the earliest tuberculin surveys with the more recent he says that in the former surveys there seemed to be an assumption that among the poorer classes of some European and American cities there was a very high proportion of tuberculin positives in late childhood, whereas recent figures with the intradermal test show considerably lower percentages. Is this due to differences in technique, or is it due to an actual reduction in incidence of tuberculous infection in childhood? He also suggests that these systematic tuberculin tests on children may throw light on a new clinical entity—transient and benign illness accompanying tuberculous first infection.

As a means of investigating contacts the test has been abundantly justified by the data produced. Some of the figures are astonishing. In Oslo, for instance, J. HEIMBECK [4] tested with the Pirquet reaction probationer nurses of the ages 20 to 25 entering the Ullevaal Hospital, part of which consists of beds for tuberculous patients. Of 282 nurses who began with a positive Pirquet reaction four developed tuberculosis, whilst of 299 who began their training with a negative reaction 67 became tuberculous. Some confirmation of these figures is given by E. K. GEER [5] from hospitals in the State of Minnesota where the incidence of tuberculous infection in young adults seems to be low. Since September, 1928, he has tested all incoming probationers at the Ancker Hospital in the city of St. Paul, Minnesota, 215 out of the 975 beds in the hospital being allotted to tuberculous patients. Thirty per cent. of these nurses showed positive reactions to the intradermal test on entering the training school, and practically all gave a positive before they had completed their three-year training period. The records of these nurses indicate that the negative

[1] The Value of Tuberculin Tests in Man. By P. D'Arcy Hart, M.D., M.R.C.P., Med. Research Council Spec. Rep. Series No. 164. London: H.M. Stationery Office. 1932. Pp. 136. 2*s*.
[2] THE LANCET, 1931, i., 872.
[3] Quart. Jour. Med., January, 1932, p. 49.
[4] Norsk. Mag. f. Laegevidensk., 1929, xc., 18. (Abstr. in Tubercle, 1931, xii., 327.)
[5] Arch. Int. Med., 1932, xlix., 77.

reactors at the end of the childhood period are virgin soil for adolescent infection. The investigation of R. J. MATTHEWS in Wales points in the same direction, though unfortunately, like HEIMBECK'S figures quoted above, his records were obtained with the old Pirquet test and are therefore not strictly comparable with GEER'S. Following up a number of child contacts over a period of years MATTHEWS found that in a group of 120 cases there was no tuberculosis in the tuberculin positive group, whereas in the negative group there were seven cases which subsequently developed tuberculosis, six of them on reaching adolescence. There is good evidence therefore that non-reactors cannot merely be dismissed with the verdict "all is well." Their chance of subsequently developing clinical tuberculosis is greater than that of the reactors. With regard to tuberculin as an aid to clinical diagnosis Dr. HART states that a positive response in infancy more than at any other age suggests a tuberculous infection which is likely to be progressive, and that clinical tuberculosis is, or soon will be, present, and moreover that the outlook is unfavourable. A positive reaction in children aged 2 to 5 *with symptoms* is also of significance, but, in Dr. HART'S opinion, the interpretation of the positive reaction in those over five years of age requires considerable experience and at the present stage defies dogmatism. We are not sure, however, that we should like to go all the way with him in saying that the diagnosis of tuberculosis in childhood should never be made without performing a tuberculin test unless there is clear bacteriological or pathological evidence of disease. These positive reactions, too, like the positive Wassermann, may prove actually a red herring. Dr. HART makes a valuable point when he states that in certain cutaneous conditions, such as lupus pernio and the sarcoids, a loss of tuberculin reactivity may be found, so that a positive response is evidence against the diagnosis, whilst a negative response, demonstrated by the intradermal test in maximal doses, strongly supports the diagnosis.

Although Dr. HART is both wisely conservative and scrupulously careful to avoid extravagant claims he has shown that much remains to be done. Tuberculin diagnosis is not the closed chapter it was thought to be even a decade ago. The next advance seems likely to be along laboratory lines. During the last few years important steps have been taken in standardisation, and quite recently a team investigation has been started in America which has already made important contributions in the fractional investigation of tuberculin products.

ANNOTATIONS

HOSPITAL STAFF PAYMENT.

THE question of paying the visiting medical staffs of voluntary hospitals has been advanced a stage by the issue of a report [1] by a joint committee of the British Hospitals Association and the British Medical Association. Institutional treatment, it is remarked, has largely displaced treatment in the home, and is tending to do so more completely. At the same time social status and private means are counting less as reasons why any patient should or should not go to hospital. This substitution of institutional for home treatment has restricted the private practice of consultants and specialists, who find it increasingly difficult to earn a living without raising their private fees. The narrowing of this field of private practice shows signs of bringing about a reduction in the numbers of consultants and specialists who are in a position to give gratuitous service to the voluntary hospitals—a tendency regarded as likely to increase. The effect of competition with the municipal hospitals is then considered.

Under the Local Government Act, 1929, local authorities may finance hospitals not only for patients of the poor-law category, but for the whole community. Such council hospitals might be staffed by part-time consultants as well as whole-time medical officers, both on a salaried basis. Their management might be in the hands of representative committees, and they might be associated with teaching schools. They might thus closely approximate to the voluntary hospitals while enjoying the powerful advantage of drawing their financial support from public funds. Again, such council hospitals may, in accordance with the terms of the Act of 1929, receive subventions from contributory schemes on account of treatment given to contributors. In the case of patients able but unwilling to pay for their treatment, council hospitals may recover payment by legal process. The constantly extending demand for institutional treatment, coupled with the shortage (in most areas) of hospital accommodation, makes it, in our opinion, very probable that local authorities will in the course of time contemplate ambitious schemes for the provision of beds for other than poor-law patients. But whether or not this proves to be the case, it is difficult to believe that the existence of public authority hospitals with paid whole- and part-time visiting medical staffs will not have a powerful influence in the direction of strengthening the demand for payment by the visiting medical staffs of voluntary hospitals.

The problem accordingly resolves itself into the devising of means whereby all voluntary hospital patients who can afford to do so may make a reasonable contribution towards the fees of the doctors who treat them as well as towards their maintenance while in hospital. The case of paying patients presents little difficulty, for it is self-evident that no patient whose means enable him to pay for treatment as a private person should be allowed to enter the general wards of a hospital. The provision in all suitable hospitals of a sufficient number of rooms or wards for paying patients, coupled with the strict application of a maximum means test for entry into general wards would, it is felt, have an important bearing upon the position and attitude of the visiting medical staffs. To what extent such provision is already available in the London area may be seen from a list of pay beds, revised up to the end of 1931, and just issued [2] by King Edward's Fund. The joint committee commends the matter to the attention of hospitals in which such accommodation is capable of extension or has not yet been introduced. Contributions obtained directly from patients through the efforts of almoners during or after treatment are said to constitute about 10 per cent. of the aggregate

[1] The report is signed by Lord Linlithgow (chairman); on behalf of the British Hospitals Association by Sir Arthur Stanley, Sir Harold Pink, Sir Ernest Morris, Mr. R. H. P. Orde, and Mr. Gerard T. Whiteley; on behalf of the British Medical Association by Sir Robert Bolam, Sir Richard Luce, Mr. Bishop Harman, and Dr. Alfred Cox.

[2] Geo. Barber and Son, Ltd., Furnival-street, London, E.C.4. January, 1932. 3*d*., post free.

income of all hospitals. With the increase of contributory schemes and paying wards these contributions may tend to disappear. The crux of the whole position is found in the disposal of funds made available to hospitals through contributory schemes, the term being used in its widest sense, and the committee asks two pertinent questions:

(*a*) Are members of existing contributory schemes paying at this time enough to meet the cost of their maintenance while in hospital and to provide a contribution towards the remuneration of the visiting medical staff?

(*b*) Is it practicable at this moment of unexampled financial stringency and unemployment to suggest a higher rate of contribution?

Conditions are not uniform throughout the country, experiments are still being made by individual hospitals, and a natural process of growth and adjustment is in evidence. The committee thinks it would be an error to attempt to drive the more backward regions to seek to hold back the more progressive, or to insist upon absolute uniformity between hospital and hospital. Apart from this there is general agreement on the payment to the visiting medical staff of a part of funds paid on account of in-patients and out-patients by the State, county or municipal or other public authorities, or by accident insurance companies.

The joint committee registers its conclusions: (1) that the time has come to recognise the claim of the visiting medical staffs to some share in the moneys raised for the treatment of patients in hospital, other than those provided by voluntary subscription or donation for the treatment of free patients; (2) that the ideal contributory scheme is one in which the individual shall contribute upon a basis designed to provide (*a*) cost of maintenance, (*b*) something towards the remuneration of the visiting medical staff; (3) the provision in all suitable hospitals of a sufficient number of rooms or wards for paying patients, coupled with the strict application of a maximum means test for entry into general wards.

THE GERMICIDAL POWER OF THE SKIN.

Two years ago L. Arnold and co-workers [1] came to the conclusion that the skin has some power to destroy bacteria. In further experiments on the same subject J. F. Norton and M. N. Novy [2] have closely followed the technique they described. The hands were immersed in a dilute suspension of *Bacillus prodigiosus*, and from the surface thus infected agar plates and broth tubes were inoculated after various periods. The colonies which grew were counted, and the results were carefully recorded. As might have been expected, the factors of moisture or dryness had a big influence. During the experiments it was found that the skin usually became dry in ten minutes, and whereas, in a series of 12 experiments on the dorsal surface of the hand, there were apparently no organisms left after half an hour, as many as 150 to 1000 colonies could be counted when the skin was kept moist by water vapour. Other materials, such as glass, filter-paper, and tanned hide were tested in the same way and found to give closely similar results, from which it is concluded "that these experiments fail to indicate any inherent germicidal power of living skin." Apart from their surgical and hygienic bearings these results are of dermatological interest. If moisture favours the growth of bacteria—and of this there is, of course, no doubt—it is easy to understand the part it plays in producing such conditions as washerwoman's dermatitis, tinea cruris and pedis, and the various forms of intertrigo, whether due to bacteria or fungi. This knowledge supports experimentally the value of desiccating or exsiccating remedies like spirit, powder-containing lotions, and absorbent powders. Equally significant are the evil results of too much moisture, and the occlusory effects of grease and ointments in the intertriginous type of case.

[1] Amer. Jour. Hyg., 1930, xi., 345.
[2] Amer. Jour. Pub. Health, 1931, xxi., 1117.

EUTHANASIA.

LAST October we reported [1] Dr. C. Killick Millard's presidential address to the Society of Medical Officers of Health, in which he pleaded for the legalisation of euthanasia. He has now reprinted his paper in pamphlet form,[2] together with the text of his proposed Bill and an appendix containing expressions of opinion by doctors, clergymen, and others. Sir W. Arbuthnot Lane, in a foreword, strongly supports Dr. Millard's views, and says he has met with widespread approval of them in the audiences, chiefly composed of working people, whom he has addressed. He points out, however, that it will probably be difficult to pass the Bill rapidly through the House of Commons, and the *Spectator* in an editorial article last autumn probably came even nearer the truth when it said that the Bill stood not the smallest chance of passage into law at present. Nevertheless, the drafting of a serious Bill is a good method of stimulating public discussion in a subject as important as this. Dr. Millard produces a considerable number of thoughtful opinions in favour of the legalisation of euthanasia subject to adequate safeguard, but only gives, as an example of adverse criticism, four extracts which on the face of them would not be likely to command much respect from reasonable minds. Surely a movement as novel and daring as this has evoked as weighty criticism as it has approbation? If Dr. Millard has received any expressions of adverse opinion at all comparable with the favourable ones he quotes, it would probably help his cause if he were to publish them.

THE TOURNIQUET.

IN the last issue of the *British Red Cross Quarterly Review* there is a tale with a moral to it. A woman had been knocked down by a bicycle in the street, and had a nasty cut a little above the left eye. An errand boy, who had secured a front-row position, asked anxiously: "Aren't you going to stop it with a tourniquet?" Most first-aid manuals take the tourniquet for granted, and give first-aid workers a vague and diplomatic warning about the dangers of too prolonged pressure without, as a rule, mentioning any time limit. Few, if any, authors of such manuals ignore the tourniquet altogether or to refer to it as a disreputable relic of the past, whose only habitat should be the museum. This is, however, the gist of the opinion expressed by Dr. M. Stassen in *Bruxelles-Médical* for Dec. 13th, 1931. During the past seven years he has dispensed with the tourniquet in the first-aid equipments for which he has been responsible, and among the several hundred cases of compound fractures of the limbs brought to his hospital from points up to 60 kilometres distant, he has not once had a death from hæmorrhage during transport. Belgian army surgeons formed a very bad opinion of the tourniquet during the Great War, and Dr. Stassen regards this instrument as a frequent

[1] THE LANCET, 1931, ii., 905.
[2] Euthanasia: a Plea for the Legalisation of Voluntary Euthanasia under Certain Conditions. By C. Killick Millard, M.D., D.S.c. London: C. W. Daniel Company. Pp. 44. 2*s*.

cause of traumatic shock and gas gangrene. By completely arresting the circulation in the injured limb, the tourniquet promotes infection in the crushed and torn tissues, and its removal is followed by absorption of toxic products capable of killing a patient already weakened by cold, shock, and loss of blood. At The Hague in 1931, during the sixth International Congress of Military Medicine and Pharmacy, various speakers condemned the tourniquet in unequivocal terms. Colonel Praag on this occasion declared that the medical personnel cannot be taught too much about the dangers of the application of the tourniquet which should not be permitted in any but cases of extreme urgency. In his report on the "Action du garrot sur le choc traumatique" Colonel Dante Casella was not less emphatic on the same occasion. The errand boy, it would seem, is now the staunchest friend of the tourniquet.

NEW NAMES FOR HOSPITALS.

ASCENDING for once from the practical to the romantic, the London County Council decided to improve the names of some of its hospitals by abandoning the territorial and utilitarian in favour of something more aspiring. It has, however, chosen the new designations with a nice regard for local susceptibilities which, even in London, are properly tender. Resisting the temptation to call its hospitals after their chairman or vice-chairman, it has entered the safer field of hagiology. St. Leonard of Shoreditch, St. Alphege of Greenwich, and St. Olave of Bermondsey are names which belong to local history. The comparatively unfamiliar St. Benedict will illuminate the hospital at Tooting which will thus be able to realise a spiritual relationship with the St. Benet to whom the City of London dedicated four of its ancient churches. The Hospital of St. Nicholas, beloved of fishermen, carries on the traditions of Plumstead; but St. Charles of Marylebone seems to be more picturesque than locally appropriate. Only two saints of that name appear to be known to the Churches—the Roman St. Charles Borromeo and the "Charles, King and Martyr" of the old Anglican calendar, to whom some five or six churches have been dedicated. That houses of healing should be given distinctive names suggestive of the spiritual basis of hospital work is all to the good. But it rests with the staff of these institutions to make the new designations so familiar that we shall speak, for instance, of St. Olave's without the need to mention Bermondsey.

RELIEF OF VESICAL IRRITATION.

As a remedy for painful conditions of the bladder, B. E. Greenberg and M. L. Brodny [1] have been using mineral oil sterilised in the autoclave, cooled and then heated to body temperature. Some 4 oz. of the oil are injected slowly into the bladder, and despite the fact that patients with cystitis suffer from increased frequency—partly because of their condition and partly because they are on "forced fluids"—some of it may remain there as long as a week. After its instillation the vesical irritation, tenesmus, and frequency are reduced; as the burning decreases the patient loses his fear of micturition and consents to drink the large amount of liquid required for adequate therapy. Greenberg and Brodny have used this method not only for ordinary cystitis, but also for tuberculosis of the bladder. They have, moreover, found it useful in alkaline cystitis where the bladder wall is encrusted with phosphates; the oil loosens the encrustations and cleans up the bladder. In a previous paper [2] Greenberg has advocated the use of sterile mineral oil as a medium for cystoscopy, finding that with irritated bladders far better distension could be obtained with oil than with boric lotion. Furthermore, the intense discomfort which often follows cystoscopy in such cases was prevented. Finally, the oil was found to encourage hæmostasis so that cystoscopy could be undertaken in the presence of bleeding that would otherwise have obscured the view. The method is a simple one, and these claims are so striking that it certainly deserves a trial.

[1] New Eng. Jour. Med., Jan. 21st, p. 114.

PROBLEMS OF RESPIRATORY DISEASE.

RECENTLY we quoted observations on draughts and colds published by Winslow and Greenburg in the January number of the *American Journal of Hygiene*. The same issue contains several other papers on various aspects of respiratory disease. Janet M. Bourn reports a further study of the occurrence of *Hæmophilus influenzæ* (Pfeiffer's bacillus) in Baltimore, based upon 2600 nasopharyngeal cultures. The bacillus was isolated with no greater frequency from cases of respiratory disease than from healthy persons. In addition, during 1928–1929 a widespread epidemic of influenza took place. The incidence of *H. influenzæ* was no higher in these epidemic months than in the following non-epidemic year, a result in agreement with the conclusions of the report published by the Ministry of Health in 1930 on the nasopharyngeal flora of a group of the Manchester population.[3] On the other hand, in the English study there was no evidence that the onset of a cold was associated with any characteristic change in the nasopharyngeal flora. In Bourn's investigation a more detailed study of cases of upper respiratory disease (which included influenza and other symptoms besides the common cold) showed a higher frequency of the bacillus amongst those with more extensive local symptoms. For example, the bacillus was found in 28 per cent. of cases with cough and in only 16 per cent. of cases without cough, a difference which is unlikely to have arisen by chance. This difference and a tendency to a greater frequency late in the attack in all cases support the general belief that this particular bacillus is of importance as a secondary invader. Frequent examinations showed that practically all persons of the classes studied harboured *H. influenzæ* at some time during the two years.

With regard to the pneumococcus, Selma M. Shultz found the duration of the carrier state amongst 35 children convalescent from pneumonia to vary between 2 and 152 days. She considers that the convalescing child, possibly carrying the pathogen in its throat for such long periods, may well be considered a potential source of respiratory diseases in others. For 21 children with pneumococcal pneumonia she endeavoured to measure the incidence of pneumococcus carriers amongst contacts. Naturally the search had to be limited to the most intimate contacts, and amongst these (86 in number) 13 per cent. produced the same type of pneumococcus as that isolated from the pneumonia patient. No significant variation was apparent in the virulence of the bacillus isolated from the cases and the corresponding carriers.

In a third paper W. E. Brown gives the results

[2] Ibid., 1931, cciv., 1366.

[3] Min. of Health Rep. on Pub. Health and Med. Subjects, No. 58. London, 1930. See THE LANCET, 1930, ii., 262.

of an experiment in the prevention of the common cold by means of a vaccine. In the experimental group were 80 medical students, and in the control group were 82. He concludes that the statistical results of the experiment show little, if any, advantage to the experimental group. The mean number of colds suffered by the experimental group (over seven months) was 1·85, with a mean duration of 7·4 days, while the corresponding figures for the control group were 2·17 and 7·6. These figures do not differ significantly, but on the other hand the estimated average duration of colds during the previous years amongst the experimental group was 10·5 days, while the same average for the control group was 7·6 days. This suggests that some improvement may have taken place amongst the experimental group, but it also points to the weakness of the investigation. Any method of investigation that allows the experimental group to be unduly weighted with "susceptibles," as is here apparent, must be unsatisfactory. To prove that the groups are comparable with regard to such factors as the amount of water (and/or alcohol), tobacco, chewing-gum, and ice-cream they consume, the time when they go to bed, and the number of baths they take weekly (to mention but a few for which this paper provides statistics), seems an idle refinement in the face of such a fundamental difference with regard to susceptibility as is suggested by the previous histories of colds.

ACCIDENTS AND HOSPITAL CONTRIBUTORY SCHEMES.

MOST of the annual reports and meetings of the provincial hospitals which come thick and fast at this season are concerned with the burden placed upon them by road accidents. In a recent broadcast speech Lord Moynihan stated that Leeds Infirmary is losing £2000 a year in this way; Bradford Infirmary last year spent £600 more than it recovered upon such cases; Southport Infirmary lost £373; the Devon and Exeter Hospital complains that the suggested uniform charge of seven and sixpence a day for the treatment of victims of road accidents is "totally insufficient." In every part of the country the story is similar, and in some places contributory schemes are becoming inadequate to meet the swollen expenses, in which motor accidents are one of the items. Birmingham has already raised its contribution; Sheffield is thinking of doing likewise; and the Merseyside Hospitals Council is seeking to widen its scheme by, among other means, appealing for those annual subscribers who in most places show a tendency to fall away. Just now Liverpool is having a rather unhappy experience. Last year its port sanitary and hospitals committee spent nearly £37,000 in excess of the estimates, almost the whole of which was attributable to capital expenditure upon transferred institutions to bring them into line with the modern type of hospital. Meanwhile Liverpool, undaunted, is hastening the completion of its new Samaritan Hospital for Women which is to cost about £150,000.

Contributory schemes, under various names, are becoming increasingly important to every hospital fed by them, and several new ones are being launched. An outstanding example of success is the Leeds Hospital Fund which last year increased its income by £10,500; it now exceeds £56,000. In ten years the workpeople's hospital committee of Gloucestershire Royal Infirmary has raised just under £100,000; last year the Torbay Hospital contributory scheme collected over £7000; in 1931 the Exmouth and District Hospital League increased its membership by 1537; the Newton Abbot contributory scheme, with over 5000 members, brought in £1500. In view of these and similar figures there is small wonder that other hospitals of varying degrees of importance have decided to establish contributory schemes. Thus Walsall General Hospital, the scene in its early cottage hospital days of the devoted labours of "Sister Dora," sees no other way of making necessary enlargements, and it is proposed to make a reciprocal arrangement for treatment of its members with the neighbouring Wolverhampton Royal Hospital which has recently established a highly successful scheme with a large basis of miners. Simultaneously Goole is considering a scheme that would enable its Bartholomew Hospital, in return for a contribution of twopence a week, to give free treatment to persons whose weekly income is under £5, with special arrangements for the neighbouring villages. Since Wolverhampton has 210 beds, Walsall 100, and Goole 24, the advantages of a contributory scheme are seen to appeal to hospitals of very varying dimensions.

DISTRICT NURSING IN LONDON.

IN her survey of district nursing in London, published by the London County Council last year, Dr. Margaret Hogarth paid a remarkable tribute to the thoroughness with which care of the sick poor in their homes is organised. "I was impressed," she wrote, "with the tremendous amount of individual and collective voluntary effort expended in securing for the poorer classes a free nursing service of the highest quality obtainable. This service is never denied to anyone who cannot pay for the services of a private nurse. . . . The qualifications of the nurses employed are the best obtainable in the nursing profession." Much of the responsibility for coördinating the work of the different bodies engaged in domiciliary nursing falls upon the Central Council for District Nursing, a voluntary organisation founded for this purpose in 1914. Nursing care for the sick poor of London in their homes is provided in three ways: by district nursing associations, either independent or affiliated to the Queen's Institute; by nurses attached to the nursing branch of the Ranyard Mission; and by a few parish nurses. It speaks highly for the work of the Central Council that Dr. Hogarth found only one part of London—the new Grove Park Estate—uncovered by the organisations now at work; and even there steps were being taken to provide a nurse. The changes in administration following the enforcement of the Local Government Act of 1929 could not fail to affect the position of the Central Council to some extent, and at the end of 1929 a deputation was sent before the London County Council to explain the Central Council's work and to offer their experience and machinery for the distribution of any future subsidy which might be made to district nursing in place of the inadequate and unequal grants previously given by the boards of guardians. The annual report of the Central Council for the year 1931 tells us that a committee was formed by the London County Council to consider the question of grants, but that no definite conclusion has so far been reached. The decision can hardly fail to be one of grave importance to district nursing organisations, since the economic crisis makes it increasingly difficult for them to obtain financial assistance. It is significant of their position that the City Parochial Foundation in making

their last contribution to the Central Council intimated that the trustees would not be disposed to continue their grants when the L.C.C. had fully developed its powers under the Local Government Act.

Upon the Central Council has fallen the duty of revising and republishing the directory of district nursing and street list for London; the 1918 edition of the directory is now out of date and its value as a reference book makes republication especially desirable from the point of view of hospital almoners, social workers, and general practitioners in the poorer districts of London. It also falls to the Central Council to award bursaries for midwifery training, to which purpose a grant from the London Parochial Charities is directed. Since this special contribution was first made, 36 nurses have been enabled to obtain the C.M.B. certificate. The desirability of extending these bursaries to cover post-certificate courses has recently been discussed, and it has been agreed that applications should be considered for such part of the cost of post-certificate courses as is not met by the Ministry of Health, provided that the candidate will agree to undertake midwifery in London for not less than a year.

A simplification of statistics and accounts is likely to follow the issue of a standard form by the Central Council for circulation among the bodies which come under its organisation. The different modes of presenting income and expenditure accounts made comparison, and the allocation of equitable grants, extremely difficult, and the issue of a standard form is another example of administrative value of the Central Council. District nursing, as Dr. Hogarth pointed out, calls for altruism and a high conception of service in the woman who takes it up, and such devotion, she believes, should not be exploited, even in the interest of the general public. THE LANCET Commission on Nursing noted the valiant efforts of the Nursing Association to obtain funds, and the manifest need for the services of district nurses, but stated their conviction that the pay offered in this field is inadequate. It is to be hoped that these views will receive full recognition in any scheme for reconstruction of the district nursing system.

A SURVEY OF EPIDEMIC ENCEPHALITIS.

THE second report [1] of the Matheson Commission for the study of Epidemic Encephalitis, which has just appeared, is arranged on the same plan as the first,[2] but on a more modest scale. Of its 155 pages considerably more than half are devoted to a continuation of the bibliography of the subject up to the end of 1929. This part of the report should be of great value for purposes of reference. The remainder consists of brief résumés of recent communications grouped under the headings Ætiology, Other Types of Encephalitis, Treatment and Epidemiology. In the second of these is given a table of incidence of post-vaccinal encephalitis in different countries, so far as figures have been available for the purpose. The chapter on treatment aims at recording every drug that has been tried, however rarely or unsuccessfully. Many of the entries might have been omitted without material loss; they serve, however, to indicate the pitfalls of drug treatment in a disease so little amenable to drugs at all. The final chapter contains a brief account of the outbreak of the disease in Japan in 1929. So far the Matheson Commission has limited its reports to the collation and the review of outside work. Since the summer of 1929 however it has been actively engaged in working on material provided at the Columbia University neurological institute, where more than 500 patients, adults and children, have been studied in relation to their response to the vaccines of Rosenow, Levaditi, and Gay. The results, to be published later, will be awaited with interest.

[1] New York: Columbia University Press. Pp. 155. $1.50.
[2] See THE LANCET, 1929, ii., 1267.

MEDICAL CARE IN INDUSTRY.

A PAPER on the Medical Care of Workers in Industry was read by Dr. N. Howard Mummery to the Epidemiological Section of the Royal Society of Medicine on Friday, Jan. 22nd. The worker, said Dr. Mummery, is to-day so carefully safeguarded that he is in danger of losing his independence and his foresight for himself and his family, and this largely at the expense of the employer of his labour, who is being held responsible for every loss of health, whether due to the employment of the worker or not. The employer is being helped by the Industrial Health Research Board, by the Home Office, and others, but wants good practitioners to assist him in the choice of his workmen in their vocational selection, in preventing disease by better hygiene, in reducing absenteeism, and increasing output. In this work the large factories already employ over 1000 doctors and 2000 nurses, working under their direction. Where Dr. Mummery works absenteeism decreases, he said, from year to year; there are 3324 men, 1461 women, and in 1930 there were 1564 male absentees from accidents and illnesses, 1601 female—a higher proportion of women—but the men were longer absent, averaging 14·12 days, the women 11·06. There was most absenteeism in January, but after that in summer months. Accidents and illnesses in frequency varied together; the reduction, year by year, he ascribed to better immediate treatment of injuries, good provision of hospital and convalescent treatment when required. The most numerous class of illnesses were colds and influenza. Dr. Mummery appealed to practitioners increasingly to devote their attention to the interesting work of assisting industry.

BY an Order of the Committee of Privy Council, Mr. W. S. Morrison, M.P., has been appointed a member of the Medical Research Council on the retirement of Major A. G. Church.

Dr. W. G. Savage has accepted an invitation to go to Boston to deliver the Sedgwick memorial lecture for 1932. He is the first Englishman to hold this lectureship in the nine years since its foundation.

Prof. Grey Turner's address on the Paget Tradition, to which we alluded last week (p. 468), was delivered on the occasion of the centenary meeting of the Massachusetts Medical Society. The Bigelow medal was awarded him by the Boston Surgical Society, and the lecture he gave on that occasion was entitled Some Experiences in the Surgery of the Œsophagus.

DEEP THERAPY AT NOTTINGHAM.—The Duke of Portland last week inaugurated the new deep X ray therapy apparatus which at a cost of £2500 has been provided to Nottingham General Hospital by the Nottinghamshire council of the British Empire Cancer Campaign.

SPECIAL ARTICLES

POLIOMYELO-ENCEPHALITIS
IN VICTORIA (1925–1931).
TREATMENT BY HUMAN IMMUNE SERUM.

BY JEAN MACNAMARA, M.D. MELB.,
HON. MEDICAL OFFICER TO THE PHYSIOTHERAPY DEPARTMENT, CHILDREN'S HOSPITAL, MELBOURNE; CONSULTANT TO THE VICTORIAN COMMITTEE FOR POLIOMYELITIS, APRIL, 1925, TO AUGUST, 1931;
AND
F. G. MORGAN, M.B. MELB.,
DIRECTOR, LABORATORIES DIVISION, COMMONWEALTH DEPARTMENT OF HEALTH, AUSTRALIA.

(*Concluded from p. 472.*)

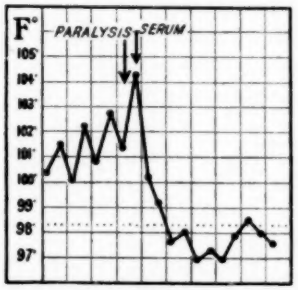
Results of Serum Therapy.

DURING the period under review serum has been administered to 260 patients. When a patient has sought treatment within 24 hours of the advent of paralysis, if fever persisted an intravenous injection of 40–60 c.cm. of serum has been given. The usual but not invariable sequence has been an immediate fall of temperature and arrest of paralysis at the stage existing when the injection was made (Fig 4).

During the months November, 1930, to July, 1931, an epidemic of 270 cases occurred. This excited great public interest. In Melbourne, where most of the cases were, families are not large; children are valuable. The press, authorities of schools, kindergartens, infant welfare centres and similar organisations, united in impressing upon parents the necessity for early diagnosis. Hundreds of children, on the slightest evidence of malaise, were taken to their doctors or to the out-patient departments of public hospitals because the parents feared paralysis. The general practitioners, many of whom had seen cases in previous years, had special lectures and demonstrations, and watched every patient suffering from indefinite illness very closely for the development of a spine sign or other warning symptoms. While it is possible that a few cases were missed, the widespread interest of public and profession makes one confident that the number of sick children not attended by a member of the profession during that period was negligible.

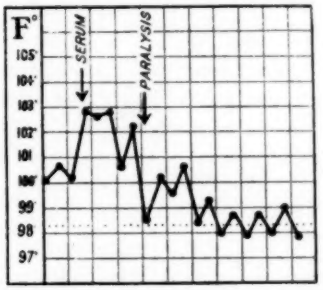
It is admitted that the only accurate method to assess the results of any therapy in preparalytic poliomyelitis would be the treatment of alternate cases, leaving the remainder as controls. In Australia this could not be done. Of the 270 cases of the epidemic, 254 were seen by one of us (J. M.), who was responsible for the after-treatment of the majority of paralysed cases by means of splinting and muscle re-education. It appeared, therefore, that an opportunity was presented to compare the cases treated with serum in the preparalytic stage with the remainder of the cases of the same epidemic.

FIG. 4.



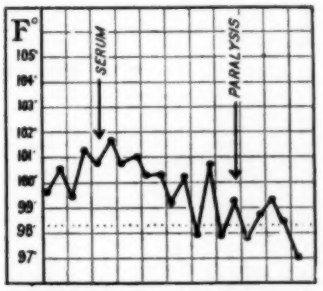
Serum given within 24 hours of paralysis. Satisfactory response.

FIG. 5.



Unsatisfactory response to serum. No second injection was given. Serious paralysis developed.

FIG. 6.



Unsatisfactory response to serum. No second dose was given. Paralysis developed.

STANDARDS OF DISABILITY.

The scheme and standards of the Harvard Infantile Paralysis Commission, as quoted in the article published by Aycock in 1929,[18] were adopted in measuring the degree of paralysis of the cases examined. The scheme may be quoted here:—

"Five degrees of paralysis are distinguished, depending on the degree of strength shown in the execution of the normal arc of motion with which the particular muscle group is concerned.

"*Good:* muscle is able to perform its function against gravity and some outside resistance.

"*Fair:* muscle is able to perform its function against gravity, but is not able to overcome any outside resistance to the movement.

"*Poor:* muscle is able to perform all or part of its normal arc of movement, but is unable to perform it against gravity.

"*Trace:* no movement is possible, but tendon can be felt to tighten on attempted motion.

"*Gone:* no response can be felt in the muscle or tendon on attempted motion.

"For purposes of tabulation and comparison of different groups of cases, arbitrary numerical values have been assigned to the different degrees of paralysis: good, 1; fair, 2; poor, 3; trace, 4; gone, 5. Muscle examination consists in tests of 106 separate muscle groups, only a few, such as palatal or facial muscles, not being subject to test for degree of paralysis. Paralysis of these muscle groups is uniformly designated as 1. According to this system of grading, complete paralysis of all the muscle groups which are tested would be represented by the figure 530 (106 muscle groups × 5). The numerical values assigned to the different degrees of paralysis do not necessarily represent the actual degree of disability, since a complete paralysis of certain minor groups may not cause as much disability as a partial paralysis of more important groups. However, it comprises a uniform method, and in so far as the number of muscle groups involved and the degree of paralysis are concerned, it is regarded as fairly accurate."

ASSESSMENT OF RESULTS.

In July and August, 1931, muscle examinations were carried out on 88 preparalytic serum-treated cases and 137 of the remainder. The delay between the onset of the disease and muscle examination favoured the controls, for the interval between onset and test averaged 8·5 weeks for the treated cases and

TABLE III.—*Synopsis of Cases Treated in the Preparalytic Stage by Human Immune Serum.*

Number.	Age and sex.	(1) Date of onset. (2) Date of treatment.	Degrees of temperature.	Spine sign.	Headache.	Clinical details.	Cerebro-spinal fluid findings.				Serum in c.cm.		Date of muscle examination.	Amount of paralysis.					
							Cells.	Type of cells.	Globulin	Chlor.§	First dose.	Second dose.		Total.	Good.	Fair.	Poor.	Trace.	Absent.
1	5 M.	12/3/26 13/3/26	100	††	†	Pain in legs and behind knees.	200	lm.	–	–	20 it. 20 iv.	12 iv.	12/4/26	0	0	0	0	0	0
2	4 M.	26/11/26 27/11/26	100	††	†	Pain in legs, diplopia.	30	lm.	–	–	60 iv.	..	8/2/27	2	2	0	0	0	0
3	10 M.	16/4/29 19/4/29	100·4	††	†	Drowsiness, sweating; three others paralysed 17/4/29.	..	..	..	..	20 iv.	..	12/5/29	0	0	0	0	0	0
4	4 M.	5/1/28 6/1/28	99	††	–	Brother paralysed; drowsiness.	35	30% pm. 70% lm.	–	–	10 iv. 20 im.	..	25/1/28	0	0	0	0	0	0
5	4 M.	28/1/28 29/1/28	99·4	†	†	Pain in leg, drowsiness.	280	20% pm. 80% lm.	†	..	40 im. 10 it.	..	21/2/28	1	1	0	0	0	0
6	6 M.	6/2/28 10/2/28	105	††††	†	Pain in legs, Amoss's sign, retention, photophobia; 15 hours' delay between puncture and serum.	45	lm.	††	750	50 iv.	30 it. 30 iv.	12/5/28	61	0	0	15	16	30
7	13 M.	6/2/28 15/2/28	104	†	†	Pains in limbs, vomiting.	180	40% pm. 60% lm.	†	..	15 it. 105 iv.	..	7/3/28	0	0	0	0	0	0
8	2 M.	12/2/28 20/2/28	101	††	–	Photophobia, drowsiness, retention.	26	lm.	†	..	10 it. 40 im.	10 im.	23/3/28	0	0	0	0	0	0
9	8 F.	23/2/28 24/2/28	99·4	††	†	Photophobia, pains in legs, hyperæsthesia.	113	28% lm. 72% pm.	††	755	30 iv. 20 im.	20 it. 20 iv.	12/4/28	5	5	0	0	0	0
				††††	††		390	50% lm. 50% pm.	††			30 im.							
10	3 F.	24/2/28 27/2/28	101	†††	†	Photophobia, drowsiness, hyperæsthesia; 36 hours' delay between puncture and serum.	..	† lm. pm.	†	760	15 it. 45 iv.	..	1/12/28	160	0	2	6	12	140
11	12 F.	2/3/28 4/3/28	103	†††	†	Brother died; paralysed 1/3/28. Pain in back.	331	† lm.	†	710	18 it. 45 iv. 5 im.	..	4/4/28	0	0	0	0	0	0
12	5 M.	16/3/28 18/3/28	101	†	†	Drowsiness, photophobia, absent jerk.	150	90% lm. 10% pm.	†	740	19 it. 29 iv. 12 im.	20 it. 20 iv.	4/4/28	3	3	0	0	0	0
13	7 F.	16/3/28 19/3/28	101·8	†	†	Pain in legs and neck, drowsiness.	250	8% pm. 92% lm.	–	–	20 it. 20 im.	20 iv.	12/4/28	0	0	0	0	0	0
14	7 M.	16/3/28 19/3/28	100	†††	†	Pain in legs, Amoss's sign.	90	† lm.	†	740	30 it. 30 iv.	..	5/5/28	2	2	0	0	0	0
15	7 F.	31/3/28 2/4/28	101	††	†	Contact with 16, 17, 18. Vomiting, pains in legs, tremor of hands.	..	† lm.	–	–	10 it. 10 im.	..	30/4/28	0	0	0	0	0	0
16	6 F.	7/4/28 9/4/28	101	††	†	Contact with 15, 17, 18. Tremor of hands, malaise, hyperæsthesia.	..	† lm.	–	–	10 it. 10 im.	10 it. 30 iv.	30/4/28	1	1	0	0	0	0
17	7 M.	17/4/28 18/4/28	102	††	†	Contact with 15, 16, 18. Tremor of hands, pain in back and neck.	150	† lm.	–	–	10 it. 30 iv.	..	30/4/28	0	0	0	0	0	0
18	1 M.	9/5/28 10/5/28	101	††	–	Contact with 15, 16, 17. Vomiting, drowsy, tremor.	20	† lm.	†	..	9 it. 30 im.	..	30/4/28	0	0	0	0	0	0
19	12 F.	12/4/28 14/4/28	99	††	†	Vomiting, drowsy, pain in neck and back.	180	lm.	†	760	30 it. 30 iv.	30 iv. 30 im.	15/5/28	0	0	0	0	0	0
20	8 M.	7/5/28 10/5/28	101	†††	†	Delirium, pain in neck and back; 12 hours' delay between puncture and serum.	100	lm.	†	720	10 it. 5 im.	..	..	Death eight hours later.					
21	11 F.	18/5/28 18/5/28	99	†	†	Pain in back and limbs. Sister of 20.	180	lm. pm.	†	720	11 it. 14 im.	..	18/6/28	0	0	0	0	0	0
22	5 M.	25/5/28 26/5/28	102	††	†	Drowsy, Amoss's sign, hamstring tenderness.	120	5% pm. 95% lm.	†	715	33 it. 47 iv.	30 im. 30 im.	17/6/28	3	3	0	0	0	0
23	8 F.	19/2/28 23/2/28	102	†††	†	Photophobia, pain in neck, vomiting.	90	lm.	†	720	15 it. 45 iv.	30 iv.	20/4/28	2	0	2	0	0	0
24	37 F.	7/6/28 11/6/28	100·4	††	††	Drowsy, vomiting, photophobia, pain in back and neck.	8*	lm.	†	720	10 it. 20 iv. 90 im.	50 im. 40 im.	7/7/28	7	7	0	0	0	0
25	3 M.	17/6/28 22/6/28	102	†††	††	Photophobia, drowsiness, opisthotonus.	20	lm.	–	720	15 it. 40 im.	..	7/11/28	0	0	0	0	0	0
26	2 F.	7/1/29 12/1/29	100	††	†	Irritable, falling over.	8*	lm.	–	760	26 it. 44 iv.	..	8/4/29	4	2	2	0	0	0
27	6 M.	18/11/28 20/11/28	105	††	†	Delirium, drowsiness, photophobia, vomiting.	200	60% lm. 40% pm.	..	..	18 it. 42 iv.	40 iv.	5/12/28	0	0	0	0	0	0
28	4 F.	18/1/29 23/1/29	101	††	†	Irritable, delirium, Amoss's sign. Brother paralysed 17/1/29.	5	lm.	†	785	26 it. 64 iv.	..	23/3/29	0	0	0	0	0	0
29	4 F.	26/1/29 28/1/29	101·2	†††	†	Vomiting, drowsiness, retention, pain in back and neck.	65	57% lm. 43% pm.	†	720	90 iv.	30 it. 8 im.	20/2/29	1	1	0	0	0	0
30	4 M.	18/2/29 26/2/29	101	††	†	Photophobia, pains in limbs, vomiting.	276	lm. pm.	†	710	17 it. 32 iv.	20 it.	1/4/29	0	0	0	0	0	0
31	4 M.	27/2/29 29/2/29	102	††	†	Vomiting, drowsy.	180	65% lm. 35% pm.	†	730	15 it. 25 im.	20 it. 10 iv.	10/3/29	0	0	0	0	0	0
32	8 M.	20/2/29 25/2/29	102	†††	†	Vomiting, pains in back, retention, toxæmia.	40	lm.	†	731	15 it. 25 iv.	..	10/3/29	0	0	0	0	0	0
33	7 F.	7/3/29 9/3/29	101	††	†	Pains in back, drowsiness.	70	lm.	†	..	10 it.	40 iv. 10 it. 10 iv.	17/6/29	3	3	0	0	0	0
34	10 M.	11/3/29 16/3/29	101·4	†††	†	Amoss's sign, drowsiness.	40	lm.	†	..	9 it. 50 iv.	30 iv.	7/6/29	1	1	0	0	0	0
35	5 M.	6/3/29 16/3/29	99·4	†	†	"Influenza 10 days before." Brother paralysed (fatal).	27	lm. pm.	†	730	10 iv. 10 iv. 10 im.	..	10/6/29	0	0	0	0	0	0
36	8 M.	14/4/29 17/4/29	103·6	†	†	Vomiting, drowsiness, abdominal pain.	120	lm. pm.	†	..	10 it.	..	7/4/29	0	0	0	0	0	0
37	9 M.	23/2/29 25/2/29	102	††	†	Photophobia, pain in limbs, drowsiness.	60	lm.	–	..	30 it.	..	24/3/29	0	0	0	0	0	0
38	1 M.	11/3/29 13/3/29	102	†††	–	Photophobia, drowsiness, vomiting.	40	lm.	†	..	10 it. 30 im.	..	8/4/29	0	0	0	0	0	0
39	5 F.	15/3/29 23/3/29	99·6	††	†	Irritable, pain in neck and back, hyperæsthesia.	36	lm.	†	700	12 it. 48 iv.	..	1/6/29	0	0	0	0	0	0
40	5 M.	16/3/29 23/3/29	101·6	†††	†	Irritable, drowsiness, Amoss's sign, hyperæsthesia.	150	lm.	††	..	20 it. 20 iv.	30 iv. 30 iv.	7/6/29	3	3	0	0	0	0

§ Chlorides in mg. per 100 c.cm. * Examination delayed.

TABLE III.—*Synopsis of Cases Treated in the Preparalytic Stage by Human Immune Serum—(continued).*

Number.	Age and sex.	(1) Date of onset. (2) Date of treatment.	Degrees of temperature.	Spine sign.	Headache.	Clinical details.	Cerebro-spinal fluid findings.				Serum in c.cm.		Date of muscle examination.	Amount of paralysis.					
							Cells.	Type of cells.	Globulin	Chlor.§	First dose.	Second dose.		Total.	Good.	Fair.	Poor.	Trace.	Absent.
41	5 F.	16/3/29 24/3/29	100	†††	†	Epistaxis, pain in neck, back, limbs; 20 hours' delay between puncture and serum.	90	lm.	..	760	30 iv.	..	1/12/31	30	9	12	9	0	0
42	9 M.	4/10/29 7/10/29	100	††	–	Stiffness in limbs, hyperæsthesia, opisthotonus.	(bs.) p.	..	..	..	40 iv.	40 iv. 40 it.	3/12/29	0	0	0	0	0	0
43	4 M.	7/10/29 14/10/29	100	††	–	Drowsiness, pain in neck and limbs.	175	lm.	..	..	25 it. 5 im.	..	24/12/29	3	3	0	0	0	0
44	6 F.	2/2/30 5/2/30	101	†	†	Vomiting, drowsiness.	†	lm.	†	..	20 it. 50 iv.	..	1/6/30	0	0	0	0	0	0
45	4 M.	5/2/30 7/2/30	101	†	†	Vomiting, drowsiness.	†	lm. pm.	†	..	20 it. 40 iv.	..	3/5/30	0	0	0	0	0	0
46	3 F.	4/2/30 18/2/30	100	††	†	Vomiting, pain in neck, back, and knees.	20	lm.	†	750	15 it. 35 im.	..	1/4/30	8	4	4	0	0	0
47	19 M.	8/2/30 10/2/30	102	††	†	Photophobia, pain in neck, retention delay after puncture.	120	lm. pm.	†	720	20 it. 70 iv.	..	1/6/30	0	0	0	0	0	0
48	2 F.	1/3/30 5/3/30	101·6	†	†	Irritable, drowsiness, constipation.	40	lm.	†	..	30 im.	..	8/8/31	94	13	20	45	16	0
49	9 M.	1/3/30 6/3/30	100	††	†	Dromedary onset, Amoss's sign, pain in limbs, vomiting.	20	lm.	†	750	20 it. 30 iv.	..	1/6/30	0	0	0	0	0	0
50	2 M.	14/5/30 17/5/30	100	†	†	Hyperæsthesia, vomiting, drowsy.	100	80% lm. 20% pm.	†	720	20 it. 70 iv.	..	1/9/30	2	0	2	0	0	0
51	7 M.	11/6/30 16/6/30	104	†††	†	Vomiting, drowsiness, diplopia.	370	60% lm 40% pm.	†	..	40 iv.	..	12/8/30	2	2	0	0	0	0
52	4 M.	26/7/30 2/8/30	100	††	–	Hyperæsthesia, delirium, tremor, pain in limbs.	150	lm.	†	750	30 it. 30 iv.	..	23/8/30	0	0	0	0	0	0
53	5 F.	1/8/30 6/8/30	100·6	††	–	Drowsiness, pain in neck.	200	lm.	†	..	20 it. 20 iv.	30 iv.	1/10/30	0	0	0	0	0	0
54	7 M.	29/8/30 3/9/30	101·4	†††	†	Tremor, photophobia, nystagmus, drowsiness.	45	lm. pm.	†	720	20 it. 40 iv.	..	1/1/31	2	2	0	0	0	0
55	8 F.	10/9/30 13/9/30	102	††	†	Delirium, vomiting, drowsiness.	90	lm.	†	..	20 it. 30 iv.	..	4/3/31	0	0	0	0	0	0
56	5 F.	18/9/30 19/9/30	101	†	–	Delirium, vomiting, loss of speech.	50	lm.	†	730	20 it. 40 iv.	..	1/10/30	0	0	0	0	0	0
57	13 F.	19/10/30 20/10/30	101	††	†	Pain in neck and back, tremor of hands.	†	lm.	†	..	10 it. 40 iv.	60 iv.	20/11/30	0	0	0	0	0	0
58	3 M.	3/11/30 4/11/30	101	††	†	Vomiting, drowsiness, tremor of hands.	110	85% lm. 15% pm.	†	750	29 it. 31 im.	..	30/11/30	0	0	0	0	0	0
59	4 M.	16/11/30 18/11/30	103	†††	†	Vomiting, diplopia, tremor, drowsiness.	150	lm.	†	..	15 it. 35 iv.	..	2/2/31	1	1	0	0	0	0
60	12 M.	26/11/30 23/12/30	100·4	††	†	Epistaxis, vomiting, abdominal pain.	600	lm.	..	..	30 it. 30 iv.	..	8/5/31	0	0	0	0	0	0
61	11 M.	14/12/30 18/12/30	103·8	†††	†	Delirium, pain in leg, drowsiness.	560	lm.	†	..	20 it. 40 iv.	60 iv.	25/1/31	0	0	0	0	0	0
62	3 M.	24/12/30 27/12/30	99·6	†	–	Tremor of hands, pain in limbs, Sister paralysed 25/12/30.	62	lm.	–	760	30 it. 20 iv.	..	10/1/31	0	0	0	0	0	0
63	4 M.	23/1/31 28/1/31	102	†††	†	Drowsiness, pain in neck, retention.	65	90% lm. 10% pm.	†	725	15 it. 45 iv.	..	21/6/31	2	2	0	0	0	0
64	3 M.	1/2/31 4/2/31	101	†	†	Abdominal pain, vomiting, drowsiness.	†	lm.	†	..	15 it. 15 im.	..	2/5/31	0	0	0	0	0	0
65	6 M.	5/2/31 6/2/31	101	††	†	Vomiting, drowsiness.	†	lm.	†	..	10 it. 5 iv.	20 iv.	7/4/31	0	0	0	0	0	0
66	2 M.	11/2/31 15/2/31	102·6	††	–	Drowsiness, retention.	30	lm.	..	720	20 it.	30 iv.	10/5/31	0	0	0	0	0	0
67	7 M.	16/2/31 17/2/31	101	††	†	Drowsiness, persistent vomiting.	120	lm.	..	720	24 it. 30 iv.	..	26/2/31	0	0	0	0	0	0
68	5 M.	16/2/31 18/2/31	99·8	††	–	Drowsiness, tremor of hands, facial paresis.	..	(bs.)	..	..	5 it. 25 im.	..	6/3/31	0	0	0	0	0	0
69	3 M.	9/2/31 11/2/31	100	†	–	Irritable, drowsy.	†	lm.	†	750	10 it. 50 iv.	..	25/2/31	0	0	0	0	0	0
70	17 F.	26/2/31 7/3/31	103	†††	†	Abdominal pain, diplopia, retention, vertigo.	12	lm.	†	720	60 iv.	60 iv.	1/8/31	3	1	2	0	0	0
71	8 M.	27/2/31 12/3/31	101	††	†	Dromedary onset, tremor, vomiting.	100	lm.	†	..	30 it. 30 iv.	..	27/4/31	0	0	0	0	0	0
72	5 M.	20/3/31 21/3/31	100	†	†	Brother of 71. Abdominal pain, drowsiness.	90	lm. pm.	..	..	30 it. 30 iv.	..	27/4/31	0	0	0	0	0	0
73	5 M.	10/3/31 12/3/31	101	†	†	Drowsiness, sweating, giddiness.	100	lm.	..	..	30 it. 30 iv.	..	19/3/31	0	0	0	0	0	0
74	10 F.	14/3/31 21/3/31	100	††	†	Sister paralysed 14/3/31. Malaise.	175	lm.	†	..	30 it. 30 iv.	..	4/4/31	1	1	0	0	0	0
75	5 F.	16/3/31 17/3/31	102	††	†	Vomiting, pain in neck.	200	50% lm. 50% pm.	..	..	30 it.	..	10/7/31	6	2	4	0	0	0
76	6 M.	3/3/31 4/3/31	99·8	†	†	Convulsion, pain in neck, giddiness.	20	lm.	..	720	15 it. 45 iv.	..	12/5/31	0	0	0	0	0	0
77	4 M.	20/3/31 28/3/31	101	†††	†	Irritability, tremor, retention.	†	lm.	..	..	30 it. 30 iv.	..	8/4/31	0	0	0	0	0	0
78	10 M.	1/4/31 10/4/31	99	†††	†	Dromedary onset, photophobia, severe; pains in limbs.	20	lm.	..	740	20 it. 30 iv.	..	16/6/31	2	2	0	0	0	0
79	7 M.	7/4/31 10/4/31	101	†††	†	Vomiting, drowsiness, tremor and toxæmia.	60	lm.	†	730	30 it. 30 iv.	..	16/5/31	0	0	0	0	0	0
80	5 M.	13/4/31 16/4/31	100	†	†	Drowsiness, tremor, pain in back.	50	60% lm. 40% pm.	†	..	30 it. 30 iv.	..	16/7/31	4	0	4	0	0	0
81	10 M.	15/4/31 16/4/31	102	††	†	Pain in neck, back, and in right arm.	300	32% lm. 68% pm.	†	..	30 it. 50 iv.	..	24/7/31	1	1	0	0	0	0
82	10 M.	27/4/31 29/4/31	102	††	†	Vomiting, abdominal and popliteal pain.	1100	lm. pm.	†	..	20 it. 50 iv.	..	8/5/31	0	0	0	0	0	0
83	4 F.	30/4/31 2/5/31	103	††	†	Photophobia, malaise, tremor of hands.	180	25% lm. 75% pm.	†	..	30 it. 40 iv.	440 iv.	10/5/31 6/8/31	3 1	3 1	0 0	0 0	0 0	0 0
84	9 F.	1/4/31 9/4/31	103·8	††††	†	Dromedary onset, tremor, retention, hyperæsthesia.	450	lm.	†	720	25 it. 55 iv. 30 im.	..	24/7/31	0	0	0	0	0	0
85	3 M.	2/4/31 7/4/31	101	†††	†	Dromedary onset, pain in right arm, tremor of hands.	300	lm.	†	730	30 it. 35 iv.	..	1/5/31	2	2	0	0	0	0
86	4 M.	3/4/31 7/4/31	100	†	†	Hyperæsthesia of thighs, tremor, pain in neck.	200	lm.	†	..	30 it. 30 iv.	..	18/4/31	1	1	0	0	0	0

§ Chlorides in mg. per 100 c.cm. (bs.) = blood-stained.

TABLE III.—*Synopsis of Cases Treated in the Preparalytic Stage by Human Immune Serum—(continued).*

Number.	Age and sex.	(1) Date of onset. (2) Date of treatment.	Degrees of temperature.	Spine sign.	Headache.	Clinical details.	Cerebro-spinal fluid findings.				Serum in c.cm.		Date of muscle examination.	Amount of paralysis.					
							Cells.	Type of cells.	Globulin	Chlor.	First dose.	Second dose.		Total.	Good.	Fair.	Poor.	Trace.	Absent.
87	2 M.	10/4/31 10/4/31	104	††	†	Vomiting, irritability.	16	lm.	†	750	20 it. 50 iv.	..	25/4/31	2	2	0	0	0	0
88	8 M.	10/4/31 13/4/31	100	††	†	Drowsiness, vomiting. Brother paralysed 11/4/31.	†	lm.	..	..	30 it. 30 iv.	30 iv.	3/6/31	0	0	0	0	0	0
89	4 F.	16/4/31 18/4/31	103	†††	†	Pain in abdomen and limbs.	150	lm.	..	..	20 it. 30 iv.	..	17/7/31	0	0	0	0	0	0
90	8 F.	23/4/31 26/4/31	103	††	†	Giddiness, pain in neck, loss of speech.	120	lm.	..	730	50 iv.	..	30/7/31	3	3	0	0	0	0
91	5 M.	24/3/31 27/3/31	101	†	†	Pain in neck and limbs.	180	lm.	..	..	30 it. 20 iv.	..	22/7/31	23	10	4	9	0	0
92	24 F.	25/4/31 30/4/31	100	††	—	Malaise, nystagmus. Sister paralysed 1/5/31 (fatal).	No lumbar puncture.				70 iv.	..	23/7/31	1	1	0	0	0	0
93	3 F.	26/4/31 30/4/31	103	††	†	Irritability, drowsiness.	25	pm. lm.	†	710	10 it. 50 iv.	..	8/7/31	1	1	0	0	0	0
94	4 M.	30/4/31 3/5/31	102	††	†	Vomiting, pain in legs, drowsiness.	60	pm. lm.	..	..	15 it. 15 iv.	..	1/8/31	2	2	0	0	0	0
95	3 M.	1/5/31 2/5/31	102	††	†	Vomiting, irritable, constipated.	150	70 % pm. 30 % lm.	..	..	30 it. 40 iv.	..	6/8/31	1	1	0	0	0	0
96	3 F.	2/5/31 9/5/31	101	†	—	Drowsiness, retention, irritability.	200	lm. pm.	..	..	30 it. 30 iv.	..	24/7/31	6	6	0	0	0	0
97	3 M.	2/5/31 7/5/31	100	†	—	Drowsiness, retention.	100	lm.	..	..	30 it. 30 iv.	..	3/8/31	0	0	0	0	0	0
98	5 M.	3/5/31 5/5/31	101	††	†	Stiffness, falling over, pain in neck.	230	lm. pm.	†	..	30 it. 40 iv.	..	1/8/31	1	1	0	0	0	0
99	1 M.	1/5/31 5/5/31	103	††	—	Vomiting, drowsiness, retention.	20	lm.	†	725	30 it. 40 iv.	..	1/8/31	0	0	0	0	0	0
100	6 F.	5/5/31 6/5/31	102·4	††	†	Drowsiness, pain in neck and back.	100	lm.	†	..	30 it. 30 im.	..	3/8/31	2	2	0	0	0	0
101	1 M.	7/5/31 11/5/31	104	††	—	Drowsiness, constipation.	580	70 % pm. 30 % lm.	†	..	30 it. 40 iv.	..	30/7/31	2	2	0	0	0	0
102	9 M.	7/5/31 9/5/31	103	†	†	Abdominal pain, tremor.	150	lm. pm.	†	..	30 it. 30 iv.	..	16/5/31 17/7/31	3 0	3 0	0 0	0 0	0 0	0 0
103	3 M.	8/5/31 12/5/31	103	†††	—	Vomiting, tremor, irritability.	480	60 % pm. 40 % lm.	†	740	30 it. 30 iv.	..	24/7/31	5	5	0	0	0	0
104	10 M.	2/5/31 4/5/31	103	††	†	Epistaxis, pain in back, tremor.	260	60 % pm. 40 % lm.	†	..	30 it. 40 iv.	..	12/7/31	1	1	0	0	0	0
105	8 M.	8/5/31 10/5/31	101·8	†††	†	Pain in calves.	†	lm.	†	..	10 it. 10 iv.	10 iv.	1/6/31	0	0	0	0	0	0
106	2 F.	10/5/31 11/5/31	102	††	—	Vomiting, drowsiness.	160	60 % pm. 40 % lm.	†	..	30 it. 40 iv.	..	24/7/31	0	0	0	0	0	0
107	9 F.	11/5/31 14/5/31	101	†††	—	Delirium, pain in right arm, tremor.	40	50 % pm. 50 % lm.	†	..	30 it. 40 iv.	..	10/7/31	0	0	0	0	0	0
108	5 F.	12/5/31 16/5/31	101	††	—	Drowsiness, pain in neck.	170	18 % pm. 82 % lm.	†	..	30 it. 40 iv.	..	17/7/31	1	1	0	0	0	0
109	4 F.	13/5/31 15/5/31	100	†††	—	Vomiting, retention, irritability.	150	92 % pm. 8 % lm.	..	..	30 it. 40 iv.	..	10/7/31	1	1	0	0	0	0
110	3 M.	13/5/31 26/5/31	102	††	†	Dromedary onset, drowsiness.	500	pm. lm.	..	..	30 it. 40 iv.	..	17/7/31	0	0	0	0	0	0
111	5 M.	13/5/31 14/5/31	101	†††	†	Abdominal pain, tremor Amoss's sign, toxæmia.	2000	90 % pm. 10 % lm.	††	..	30 it. 40 iv.	..	21/7/31	71	16	4	12	24	15
112	19 ..	11/5/31 15/5/31	101	†	†	Stiffness, pain in back and abdomen.	300	70 % pm. 30 % lm.	†	720	20 it. 60 iv.	50 iv.	3/8/31	2	2	0	0	0	0
113	2 F.	12/5/31 20/5/31	100	†	—	Dromedary onset, abdominal pain, drowsiness.	120	60 % lm. 40 % pm.	†	730	30 it. 40 iv.	..	27/7/31	0	0	0	0	0	0
114	6 M.	13/5/31 17/5/31	101·2	††	†	Irritability, drowsiness.	260	20 % lm. 80 % pm.	..	..	30 it. 40 iv.	..	30/7/31	36	24	6	6	0	0
115	6 M.	15/5/31 22/5/31	101	††	†	Dromedary onset, tremor, pain in neck.	200	40 % lm. 60 % pm.	†	..	30 it. 40 iv.	..	23/7/31	0	0	0	0	0	0
116	9 M.	16/5/31 24/5/31	100	††	†	Dizziness, pain in neck, photophobia, diplopia.	40	40 % lm. 60 % pm.	..	730	30 it. 30 iv.	..	23/7/31	1	1	0	0	0	0
117	2 F.	17/5/31 19/5/31	102	†	†	Vomiting, drowsiness.	40	25 % lm. 75 % pm.	..	..	20 it. 20 iv.	..	24/7/31	1	1	0	0	0	0
118	3 M.	14/5/31 17/5/31	101	†	†	Vomiting, pain in neck, irritability.	120	pm. lm.	..	..	30 it. 30 iv.	..	27/7/31	61	16	34	6	0	5
119	2 M.	17/5/31 18/5/31	101	††	†	Vomiting.	120	66 % lm. 33 % pm.	..	..	30 it. 40 iv.	..	17/7/31	1	1	0	0	0	0
120	3 M.	26/5/31 4/6/31	104	††	†	Drowsiness, vomiting, diarrhœa.	40	lm.	..	..	15 it. 50 iv.	..	17/7/31	2	2	0	0	0	0
121	3 F.	18/5/31 25/5/31	101	†	..	Drowsiness, vomiting, retention.	237	60 % lm. 40 % pm.	..	720	10 it. 30 iv.	..	7/6/31	5	5	0	0	0	0
122	1 M.	9/5/31 11/5/31	101	†	—	Drowsiness, diplopia.	..	(bs.)	..	..	20 it.	..	1/8/31	1	1	0	0	0	0
123	22 M.	21/5/31 27/5/31	101	†	†	Vomiting, pain in back, retention.	150	32 % lm. 68 % pm.	†	731	30 it. 70 iv.	90 iv.	1/8/31	0	0	0	0	0	0
124	2 F.	24/5/31 26/5/31	102·4	††	†	Vomiting, drowsiness.	200	50 % lm. 50 % pm.	..	..	30 it. 30 iv.	..	1/8/31	0	0	0	0	0	0
125	19 F.	24/5/31 28/5/31	99	†	†	Giddiness, stupor, nystagmus.	10	lm.	..	720	70 iv.	..	23/7/31	5	5	0	0	0	0
126	3 M.	27/5/31 28/5/31	101	†	†	Drowsiness, pain in limbs.	80	70 % pm. 30 % lm.	..	..	30 it. 50 iv.	..	23/7/31	0	0	0	0	0	0
127	24 F.	28/5/31 4/6/31	102	†††	†	Vomiting, pain in neck and shoulders.	95	54 % pm. 41 % lm.	..	..	30 it. 60 iv.	40 iv.	12/8/31	0	0	0	0	0	0
128	3 M.	2/6/31 3/6/31	100	†	†	Marked irritability, pain in head.	200	lm.	..	..	40 it. 40 iv.	..	17/7/31	0	0	0	0	0	0
129	8 M.	1/6/31 3/6/31	103	††	†	Giddiness, drowsiness, vomiting.	180	lm.	..	..	30 it. 30 iv.	..	28/7/31	0	0	0	0	0	0
130	16 M.	5/6/31 6/6/31	102	††	†	Pain in neck, giddiness.	123	100 % pm. 23 % lm.	†	720	30 it. 40 iv.	40 iv.	3/8/31	1	1	0	0	0	0
131	11 F.	20/6/31 22/6/31	102	†	†	Vomiting, pain in back and abdomen.	150	lm.	..	710	30 it. 30 iv.	..	17/7/31	0	0	0	0	0	0
132	6 M.	21/6/31 24/6/31	102	†	†	Vomiting, drowsiness, pain in neck.	250	lm.	..	..	20 it. 50 iv.	..	23/7/31	0	0	0	0	0	0
133	6 M.	24/6/31 26/6/31	101	††	†	Convulsion, drowsiness, retention	†	lm.	†	..	40 iv.	..	7/8/31	0	0	0	0	0	0
Complete series of treated cases, 1925–1931. Number 133.											Total muscle examinations (132)			653	183	104	108	68	190
											Average paralysis per case			4·9	1·39	0·78	0·8	0·5	1·42

17 weeks for the remainder. During that interval identical after-treatment had been carried out in the paralysed cases of both groups. Of 254 cases seen by one of us, 12 apparently successful preparalytic serum cases must be omitted from the series because evidence is incomplete; contamination of the cerebro-spinal fluid by blood prevented an accurate cell count. Fifteen paralysed cases of moderate and severe degree must be omitted because examinations according to this method could not be undertaken. The comparison is therefore between 76 cases treated by serum in the preparalytic stage and 151 others, while the condition of an additional 27 is known.

TABLE IV.—*Untreated Cases, November, 1930, to July, 1931.*

Number in series .. 151. Deaths .. 14. Survivors .. 137.

Details of 25 cases to demonstrate grades and extent of paralysis in this group.

Case No.	Age and sex.	(1) Date of onset. (2) Date of examination.	Amount of paralysis. Total.	Good.	Fair.	Poor.	Trace.	Absent
1	3 F.	12/11/30 18/7/31	101	4	24	33	20	20
2	3 F.	22/11/30 1/8/31	0	0	0	0	0	0
3	5 M.	22/11/30 30/11/30	Death.					
4	3 M.	17/11/30 1/8/31	32	12	20	0	0	0
5	8 F.	19/11/30 7/7/31	0	0	0	0	0	0
31	3 F.	16/1/31 22/7/31	9	5	4	0	0	0
32	4 F.	26/1/31 20/7/31	43	1	6	36	0	0
33	13 M.	1/1/31 23/7/31	97	3	8	24	52	10
34	3 M.	25/1/31 6/8/31	68	1	0	6	16	45
35	13 M.	1/1/31 17/1/31	Death.					
66	1½ M.	29/3/31 22/7/31	19	5	4	6	4	0
67	6 M.	27/3/31 6/8/31	379	9	6	57	92	215
68	2 M.	29/3/31 22/7/31	108	6	16	9	12	65
69	2 F.	29/3/31 28/7/31	57	4	6	30	12	5
70	2 M.	27/3/31 28/7/31	117	13	8	9	32	55
100	9 F.	18/4/31 20/7/31	21	11	10	0	0	0
101	20 F.	16/4/31 29/7/31	44	5	6	15	8	0
102	16 M.	13/4/31 22/7/31	83	4	24	42	8	5
103	12 F.	29/4/31 17/7/31	15	0	0	0	0	15
104	3 M.	1/4/31 14/8/31	2	2	0	0	0	0
134	4 F.	29/5/31 3/8/31	31	11	6	6	8	0
135	15 F.	3/5/31 2/8/31	16	9	4	3	0	0
136	3 M.	26/5/31 1/8/31	6	6	0	0	0	0
137	2 F.	4/5/31 14/8/31	24	8	4	12	0	0
138	8 M.	15/5/31 17/8/31	89	16	26	39	8	0
25 cases: 2 deaths, 23 survivors. Total paralysis			1341	135	182	327	272	435
Remaining 126 cases: 12 deaths, 114 survivors. Total paralysis			4784	990	945	1062	852	925
Total paralysis of complete series. 151 cases: 14 deaths, 137 survivors ..			6125	1125	1127	1389	1124	1360
Average paralysis per survivor ..			45·4	8·21	8·22	10·13	8·93	9·92

The survey, therefore, may be regarded as representative of the total case incidence of 270 cases. Details of the histories of cases treated in the preparalytic stage during this epidemic are shown in Table III. (Cases 58–133). Limitation of space prevents printing of the results of muscle examination in the complete series of untreated cases; but the records of 25 of these cases are given as a sample in Table IV., together with the summation of the findings for the complete series of untreated cases.

The comparison of degree and extent of paralysis in treated and untreated cases during the epidemic of 1930–1931 is shown in Table V.

TABLE V.—*Comparison of Treated and Untreated Cases.*

Nov., 1930, to July, 1931.	Number.	Death.	Paralysis. Total.	Good.	Fair.	Poor.	Trace.	Gone.
Untreated cases	151	14	6125	1125	1127	1389	1124	1360
Treated cases	76	0	266	133	66	33	24	20
Average paralysis per survivor, November, 1930, to July, 1931—								
Untreated cases ..	137	—	45·4	8·2	8·2	10·1	8·9	9·9
Treated cases	76	—	3·5	1·7	0·8	0·4	0·3	0·2
Complete series of treated cases, 1925–1931.. ..	133	1	653	183	104	108	68	190
Average paralysis per survivor	132	—	4·9	1·39	0·78	0·8	0·5	1·42

The prognosis for ultimate recovery of function depends upon the number of muscle groups affected to the degree connoted by the standards: poor (3), trace (4), or absent (5). If all paralyses could be classified as grades (1) good, and (2) fair, poliomyelitis would lose its terror; for muscles paralysed to either of these grades achieve recovery if after-treatment be carried out. When muscles are affected to the grades trace (4) or absent (5) recovery rarely occurs; and in those paralysed to the third degree (poor) recovery necessitates years of careful after-treatment.

Table VI. compares the treated and untreated cases from this aspect of the incidence of severe paralysis with its sequelæ of permanent crippling and loss of economic independence.

TABLE VI.

—	No.	Deaths.	No. with paralysis of Grades 3, 4, and 5.	Percentage of survivors affected in Grades 3, 4, and 5.	Case No.	Grades— 3 Poor.	4 Trace.	5 Absent.
Treated cases	76	0	4	5·2	91	9	0	0
					111	12	24	16
					114	6	0	0
					118	6	0	5
Untreated cases	151	14	88	58·2	Aver. per survivor	15·7	12·7	15·4

Failures.

Of the complete series treated (133), seven must be regarded as failures—No. 20 (the fatal case), and Numbers 6, 10, 41, 111, and 118. Cases 6, 10, 41, and 20 shared one common feature in their history—namely, *delay* between withdrawal of a large

quantity of cerebro-spinal fluid and administration of serum. With one exception (Number 47) they are the only cases in which this delay occurred. In the others, examination of the fluid has been made forthwith and serum administered within one hour of lumbar puncture. It is suggested that the failure of these cases may be due to one or both of two factors: (1) delay, elapse of hours allowing death of cells to occur before passive immunity was induced; and (2) that the actual withdrawal of large quantities of cerebro-spinal fluid during the preparalytic stage may have actually determined involvement of the cord.[15]

Case 48 was given one injection of serum—30 c.cm. intramuscularly on the third day of her illness—and although the temperature did not fall, no second dose was given. Her paralysis confirmed the lesson taught by a small series of cases treated in Tasmania with Victorian serum, that 30 c.cm. is usually an inadequate dose. The temperature charts of Cases 111 and 118 reveal the explanation for their failures (Figs. 5 and 6).

To Case 111, through confusion with cerebro-spinal meningitis, and to Case 118, through an oversight in the rush of the epidemic, no second injection was given, though the response to the first was not satisfactory. These failures, together with the paresis, which, although clearing up rapidly and completely, could be demonstrated in 60 other treated cases, proved beyond question that of the total series of 133 cases diagnosed and treated before paralysis, 66 were actually cases of poliomyelitis. The remainder, who developed no paralysis, presented the same clinical picture as this group of 66. Moreover, no other disease developed subsequently to suggest any other diagnosis for the signs and symptoms presented.

Conclusions.

1. It has been possible to diagnose poliomyelitis as manifested in Victoria between 1925–1931 in the preparalytic stage.
2. The use of human immune serum at this stage, in adequate dosage, has given excellent results as evidenced by a low mortality-rate, a low average total paralysis, and a strikingly low proportion of paralysis of the severer grades (as has been reported in 1929 by Aycock[18]).
3. Delay between withdrawal of cerebro-spinal fluid for examination and the administration of serum is to be avoided.
4. The dosage required is variable, and a second injection should be given if the response to the first is not prompt and satisfactory.
5. It is advantageous that the serum used should be free from any substances which provoke reactions.
6. The viricidal properties of the serum available for use should be repeatedly tested by inoculation of monkeys against specimens of virus obtained in different epidemics.
7. If supplies are ample, administration of serum within 24 hours of the development of paralysis to a febrile patient is justified, and is usually followed by a fall of temperature and arrest of paralysis.
8. If given later, when paralysis is stationary, serum is of no value.
9. In the "ascending" group of cases (Landry's paralysis) the administration of serum is justifiable, but there is no certainty that the spread of paralysis will be arrested.[19]
10. Adequate after-care of all potential donors—paralysed adults and children over the age of 10—in the intervals between epidemics, and personal practical interest in their struggle for recovery is the crux of any scheme for provision of serum stocks in an emergency. By these means the store of immune human serum has been adequate to all demands of Victoria and Tasmania during the six-year period.

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THE LANCET COMMISSION ON NURSING.

(*Continued from p. 475.*)

THE table of contents of the Final Report[1] of this Commission, which was published in THE LANCET of Feb. 20th, showed that the extent and the nature of the shortage of candidates for the nursing profession is carefully examined in Section IV., and the main reasons for the shortage is discussed in Section V. under the following headings: (*a*) The Gap between School and Hospital; (*b*) Pay and Prospects; (*c*) Hours of Work; (*d*) Conditions of Admission and Service; (*e*) Professional Education. The reasons for the shortage of trained staff are examined and discussed in Sections VI. and VII. Section VIII. is devoted to the staffing of non-approved hospitals, and Sections IX. and X. respectively to the special difficulties of tuberculosis and mental hospitals. In Sections XI. and XII. the Commission revert to some controversial aspects of the general problem. Section XI. is set out in full below.

Some Questions of Policy.

Certain proposals to which allusion has already been made[2] demand brief notice. These concern the advisability of:—

(*a*) Establishment of schools of nursing connected with universities.
(*b*) Replacement of five registers by one register.
(*c*) Substantial shortening of courses of training.
(*d*) Official recognition of two grades of nurses.
(*e*) Propaganda.

Most of these issues are outside our terms of reference. Changes as fundamental as (*a*), (*b*), (*c*), and (*d*) would involve considerations other than the relief of a shortage of candidates, and could not properly be urged solely with a view to relieving such shortage. Since, however, considerable stress has been laid on one or other of these proposals in some of the evidence submitted to us, it seems

[1] The Report may be obtained from THE LANCET Office, 7, Adam-street, Adelphi, W.C.2, for 2*s.* 6*d.*, post free 2*s.* 9*d.*, or through any bookseller.
[2] See THE LANCET, Feb. 20th, p. 416.

desirable to outline our reasons for making no recommendations on them.

ESTABLISHMENT OF SCHOOLS OF NURSING CONNECTED WITH UNIVERSITIES.

Nursing is essentially a craft, and as such is inseparable from the material on which it is exercised. While the endowment of schools of nursing is highly desirable (see para. 120), it can be argued that in order to maintain the practical quality which has distinguished British nursing, such schools should be attached to hospitals rather than to academic institutions of university status.

Certain members of the nursing profession specialise in branches such as administration, teaching, and dietetics, and training in such branches may be a function of a university. Diplomas in nursing for which only trained nurses are eligible are available in the universities of London and Leeds. In Leeds attendance at a three-months' course of lectures is required; in London the diploma is not an "academic" diploma—i.e., the nurse who has had the requisite experience can sit for the examination without having attended any special course. It is probable that there will be an increased demand for such diplomas in the future.

A SIMPLER FORM OF REGISTRATION.

The institution of one register with supplementary diplomas may be desirable when our hospital system is more unified, but the recruitment of nurses, particularly for the special hospitals, would not be furthered by a reorganisation of this kind at the present time. It is obvious that if a less complicated system of registration had been considered possible 12 years ago, it would have been adopted. The reasons against a simpler system which applied in 1919 are no less valid to-day. The introduction of more than one register was a measure designed to help the mental, fever, and children's hospitals to enlist probationers by offering them State recognition, and though one of these groups (mental hospitals) has taken little advantage of the opportunity offered (see para. 321), the removal of such opportunity would make it less rather than more easy for special hospitals to provide nursing care for their patients.

From the point of view of recruitment alone, it might be desirable not to abolish the Supplementary registers, but to raise the prestige of nurses on these registers by enabling them to qualify for inclusion in the General register after short additional courses. At the present time the regulations of the General Nursing Council permit nurses whose names are on any Supplementary register to be admitted to the general part of the register after only two years' training in an approved complete training school; but few such schools are willing to accept nurses on these terms, and in practice a registered mental, fever, or children's nurse usually has to start afresh as a probationer and work for three years before admission to the General register.

Widespread coöperation between different hospitals, or groups of hospitals, seems a goal unlikely to be reached in the near future for reasons quite unconnected with nursing. The fact that the voluntary hospitals of the country are separate autonomous institutions, competing with each other for support from the charitable public, and jealous of their reputations as training schools for nurses, places difficulties in the way of coöperative training. The organisation of combined training courses of reasonable length at municipal hospitals might be less formidable, since mental and fever hospitals are for the most part under Local Authorities. Few girls, however, would be willing to bind themselves to a course of training lasting for more than four years; while general hospitals would hardly agree to shorten the part of the training taken in those hospitals sufficiently to enable much time to be spent in mental, fever, or children's hospitals.

The registers are never likely to be reduced to less than three in number. The qualities and experience needed for mental nursing differ from those needed in general nursing, and a separate register for mental nurses will probably always be desirable; while a separate register for male nurses is convenient. The Supplementary parts of the register for those trained respectively in the nursing of infectious diseases and in the nursing of children come into a different category, but we do not at the present time recommend their abolition.

SUBSTANTIAL SHORTENING OF COURSES OF TRAINING

The substantial shortening of the course of training—i.e., to two years and four months—was recommended by the Rockefeller Committee in America,[3] but the proposal found little favour in that country, nor would it be likely to receive support in Great Britain. The leaders of the nursing profession in this country point to the large number of departments in which the nurse must gain experience, as a reason why no substantial shortening of the course could be contemplated. It would obviously be necessary to allocate nurse students to posts for two instead of for the usual three months in the wards and special departments, if all-round experience were to be gained, but they consider it doubtful whether a shorter experience would be sufficient to give any but the exceptional girl an insight into her duties. Those who advocate a shortened course argue that an educated woman could gain the necessary theoretical grounding, and learn nursing procedure in every branch of her work, in a shorter time than three years, provided that she was relieved of domestic work once she had become proficient in it. They hold that if the nurse is taught fundamental principles—e.g., asepsis, the avoidance of shock, the care of the skin—she should be able to apply these principles in all branches of her work without long experience in each department.

The prospect of shorter training might well attract some girls, especially those who desire ultimately to go into other branches of nursing and do not care to contemplate three years of institutional life; but there would be the disadvantage from the point of view of the hospitals that the institutions would benefit from the services of each probationer only over a shorter period. From the point of view of the rising generation of nurses the shortened training, with a bigger turnover, might bring the risk of overcrowding in the profession. There seems, however, to be little advantage in discussing in detail arguments for and against a substantial shortening of a three-year course, because though the proposal might be welcomed by prospective candidates, it is not likely to be acceptable either to the nursing profession or to the hospitals.

GRADING.

We are aware of a widespread feeling among the public and among a certain section of the medical profession that the training of a nurse at present is much too elaborate and that, at least for chronic

[3] Nursing and Nursing Education in the United States (Report of the Committee for the Study of Nursing Education). New York: The Macmillan Company, 1923, 15s., and THE LANCET, 1923, ii., 1203.

cases, a less highly equipped attendant would serve. The recognition by registration of two grades of nurses would, it is said, bring back into the profession those who have a real vocation, but whose lack of previous education, or incapacity for book-work, unfits them to pass the examinations now required of every State-registered nurse. The opponents of grading base their arguments on its dangers to (1) the nursing profession and (2) the public. They point out that the hard-won professional status of a nurse would be endangered thereby and that her economic position would also suffer, since, in private work at least, a second-grade nurse would be likely to undercut her. In this way the introduction of grading might, it is held, prejudice recruitment of the best type of girl to the profession. The danger to the public would be that a second-grade nurse might be entrusted, through ignorance of her limitations or for economic reasons, with responsibilities which she had not been trained to fulfil, and disastrous results might ensue. The strong feeling in the nursing profession that women trained for a shorter time on a much modified syllabus should not be allowed to use the honoured title of "hospital nurse" can readily be understood. That there is room, especially in hospitals not approved for training, for attendants who could attend to the physical needs of the patients and make them comfortable under the supervision of a trained nurse, is probable. It is probable also that such attendants would be appreciated in homes where help is required with chronic invalids. But the conditions of their employment on a large scale, and the methods of safeguarding the State-registered nurse, are matters which would require much consideration.

PROPAGANDA.

It has been urged upon us that some more systematic propaganda on the needs and attractions of the nursing profession is required. Evidence that the attention of girls has not been sufficiently directed to this profession has reached us from many sources. The circulation in schools of interesting and attractively printed pamphlets, the display of notices explaining the openings and methods of entering the profession, and the arrangement of visits to hospitals, would doubtless stimulate recruitment. The insertion of advertisements in journals read by girls of the age and type it is desired to enlist, the provision of lectures and the production of films illustrating the training and career of hospital nurses would also be of value.

Extensive propaganda of this kind would, however, be premature until some of the developments, to the need of which we have drawn attention, have taken place. For example, talks to schoolgirls might well be ineffectual until some means could be suggested to bridge the gap between school and hospital. On the other hand, a wide distribution of lists of hospitals approved as training schools, and of pamphlets explaining the complicated systems of training at various types of hospital, and the importance of ascertaining in advance whether the training at any given hospital leads to some form of State registration in two (as in fever hospitals), three, or four years, would be greatly appreciated. Every girl who enters a hospital without clearly understanding the conditions offered is likely to give the impression to others that the rules governing State registration are difficult to comply with; whereas the real difficulty lies in the fact that it is no one's business to explain their import to the candidate.

(To be concluded.)

SCOTLAND.

(FROM OUR OWN CORRESPONDENT.)

THE GLASGOW HOSPITALS.

EACH of the three great general hospitals of Glasgow—the Royal, the Western, and the Victoria Infirmaries—reports a heavy excess of expenditure over income in 1931. On ordinary expenditure the Royal had a deficit of £24,200, the Western of £12,500, and the Victoria of over £20,000. The position of the Royal Infirmary is especially unhappy, notwithstanding that it is managed so economically that the average annual cost per occupied bed is £120 17*s.* 6*d.*, as compared with the £177 spent in "one of the largest hospitals in England outside London." The in-patients are always in excess of the recognised bed state, which is a little under 800. Yet the average last year was a fraction over that figure, and the number has been as high as 870. Ordinary subscriptions and donations from employees fell by £5200, and there is no immediate prospect of improvement in this respect. The working deficiency was made up out of legacies which aggregated £47,000. As with practically every other hospital, road-accident cases helped to strain resources both in beds and money. There were 541 such cases under treatment for 8766 days at a cost of £3287, of which only £826 was recovered from the insurance companies. Among recent developments at the Royal Infirmary are a massage school for male students, and a medical gymnasium which has not only been of service to massage pupils but to the orthopædic department. It is now possible to give medical gymnastics and remedial exercises to patients in groups, instead of taking each individually. The hospital is negotiating for a supply of radium from the Commission, but the quantity is expected to be small.

Although the Western Infirmary also worked at a loss last year its report is, on the whole, more cheerful reading. At £70,780 its income was less by £3594, again as the result of unemployment; the expenditure was £83,300, but, taking legacies into account, receipts exceeded expenditure by nearly £7000. There is not the overcrowding which adds so greatly to the difficulties of the Royal. The average daily number of patients was 587, a little less than the bed capacity. The average stay was 17·04 days, and the average cost per patient £6 9*s.* 3*d.*, an increase of a few pence; the daily cost was 7*s.* 7*d.* There was an abundant supply of aspirant probationers, 594 applications having been received. The new ophthalmological department is now being proceeded with; the new radiological department, which has now been at work for a full year, is of great value, and the demands upon it are continually increasing. The hospital, which is the National Radium Centre for Glasgow, is receiving an increasing number of cancer cases from most parts of Scotland, and "the results in the cases of malignant disease suitable for radiation therapy are regarded as satisfactory."

Last year the Victoria Infirmary exceeded all previous figures in the number of its in-patients, which increased by 1136 to 8616. This was partly accounted for by the 448 persons treated in the paying patients' annexe which has been fully occupied since it was opened in April last. The waiting-list for the free beds is usually about 400. There were 217 in-patients and 317 out-patients suffering from motor accidents; £600 was received from the insurance companies. The ordinary income was £51,343, and

the ordinary expenditure £71,816, and after various adjustments the net deficit of £14,940 was drawn from capital account.

THE TEACHERS OF MENTAL DEFECTIVES.

The Scottish Education Department has decided to shorten the course for teachers of mentally defective children from six months to three months. At present the teaching is conducted at Jordanhill Training Centre, Glasgow. By rearranging and intensifying the instruction, the National Committee for the Training of Teachers have provided a course which, it is believed, will be as effective as the longer one. The cost of the special training will thus be reduced. It will be possible to provide a larger supply of qualified teachers for the mentally defective. At present there is a shortage. The teaching of mental tests has been elaborated in very specialised ways, and careful training of the teachers is essential if their opinion of an "intelligence quotient" is to be worth anything. On the other hand, as Prof. Cyril Burt long ago pointed out, a good teacher is of more importance than the most refined tests. The fundamental problem is how to distinguish between "backward" and "defective," and once the distinction is made, how to secure them appropriate places in the industrial world. The Scottish National Committee has taken this step after extended consideration.

MATERNITY HOSPITAL DEATH-RATES.

Dr. James Young (Edinburgh), in an address on the Organisation and Administration of Maternity Hospitals, has stated that in the Glasgow Royal Maternity Hospital during 1930 there were 119 deaths in 4469 cases (26·6 per 1000), while in the Edinburgh Royal Maternity Hospital during the same year there were 36 deaths among 2305 patients (15 per 1000). He compared these results with the record of the East End Maternity Hospital in London, which had 8890 births with 10 deaths (1·1 per 1000). He pointed out that these differences were partly due to the fact that in the case of Glasgow and Edinburgh institutions there was a high concentration of abnormal and difficult maternity cases from the large areas which they served. This, however, did not explain everything, because a comparison of the same class of case showed that in the hospital in the poor quarter of London there had been 200 cases of the toxæmias of pregnancy with no deaths, whereas in Glasgow there had been 448 such cases with 22 deaths (4·9 per cent.) and in Edinburgh 17 out of 208 cases (8·1 per cent.). The same facts were revealed in regard to a comparison of other types of complications. Dr. Young pointed out that the high death-rate in the Scottish hospitals arose from factors over which they had little control. The patients arrived at the hospital with their health and chances already jeopardised. In other words, the hospitals were handicapped in so far as the cases arrived often in such a state that little could be done. The lesson to be drawn from these comparisons was that when the domestic midwifery service and the service for the supervision of pregnancy were sound, the hospital death-rate was small. When it was inferior, this death-rate was high. The inference was that there was urgent need for a revision of our maternity methods. Another fact of importance was that our central hospitals admitted patients who were "suspect" in the sense that they had suffered damage before their admission. This class of case endangered the normal healthy women, because they were so apt to be infective. To meet this risk, the modern hospital must have a system by which the two classes of cases were rigidly segregated from one another in separate buildings under a separate staff.

IRELAND.

(FROM OUR OWN CORRESPONDENTS.)

MEDICINE IN THE NEW DÁIL.

MEDICINE is represented in the new Dáil by eight members. Mr. G. Hennessy, Dr. Myles Keogh, and Dr. T. Higgins retain their seats as members of the Cumann na nGaedheal Party; Sir James Craig has again been returned as an Independent; Dr. J. Ryan and Dr. F. Ward have held their places in the Fianna Fail Party, and they have been joined by two newcomers, Dr. J. B. Lynch and Dr. F. Humphrys. Two former members of the Fianna Fail Party, Dr. P. J. O'Dowd and Dr. J. A. Tubridy, failed to secure re-election. The total number of medical men in the Dáil is therefore the same as before.

Speculation is busy as to the effect on medical affairs of the change of Government which will take place next week. It is recognised that the outgoing Government has a good record in regard to health legislation. During their ten years of office a system of public health administration has been established, and there has been radical improvement in the condition of the local hospitals. The reforms in local administration have led to increased efficiency, and the work of the Appointments Commission is gradually improving the quality of entrants to the public medical service. A good deal remains to be done, however, for Ireland still lags behind neighbouring countries in many matters concerning health. It is hoped that the incoming Government may adopt a vigorous health policy as a line of action which would cause little strenuous opposition, and therefore be safe for a Government which rests on a narrow majority to pursue. The influence of the Labour Party, which is bound to be considerable, may be relied on to be used in this direction. Already the leaders of that party are insisting that a national housing scheme must be one of the earliest activities of the new Government. It is probable that medical treatment for insured persons and the conversion of the public medical services into a national medical service will also have the support of the Labour Party.

PRESENTATION TO PROF. SINCLAIR.

In the Great Hall of the Queen's University of Belfast on Feb. 16th a tribute of affection was paid to Prof. Thomas Sinclair when he was presented with his portrait in oils, painted by Mr. George Harcourt, R.A. Viscount Bangor presided over a large company of Prof. Sinclair's medical colleagues, former pupils, and personal friends. Sir Thomas Houston, in asking the Recorder of Belfast to make the presentation, referred to the great work which Prof. Sinclair did for the Belfast Medical School during the 37 years he occupied the chair of surgery, and which entitled him to be styled the founder of modern surgery in Ulster. In presenting the portrait Judge Thompson said that hundreds of people had been eager to be associated in this permanent memorial. The portrait will hang in the Great Hall in the years to come, for Prof. Sinclair on receiving the gift presented it to the University, and it was accepted by the Vice-Chancellor as a portrait of a beloved and honoured alumnus as well as a notable work of art. It will appear in London at this year's exhibition of the Royal Academy.

UNITED STATES OF AMERICA.

(FROM OUR OWN CORRESPONDENT.)

AUTOMOBILE ACCIDENTS IN 1931.

An impressive summation of facts and figures regarding the automobile accidents of last year is presented by the Travellers Insurance Co., of Hartford, Conn., in a brochure. The growth of automobile fatalities is shown in five-year periods; 58,552 for the period ending 1921, 98,551 for the period ending 1926, 152,732 for the period ending last year, and a row of alarmingly large question marks for the period ending 1936. These deaths are then analysed in a number of interesting ways and the accidents are described and illustrated by photographs and diagrams. One excellent photograph strikingly proves how much more visible is a pedestrian facing the headlights than the same man at the same distance, but with his back to the headlights. The exercise of personal responsibility is regarded as the fundamental prerequisite to accident prevention, and these striking illustrations, one of which shows the total of deaths and injuries from automobiles last year (1,032,000) in comparison with the populations of Nevada, Delaware, Wyoming, and the District of Columbia (1,042,000), will certainly aid in promoting responsible behaviour. The brochure will be valuable alike to those who are interested in the causes of automobile accidents and those who will prize a fine example of publicity applied to a worthy cause.

UNEMPLOYMENT AND MALNUTRITION.

When inaugurating a new service of wireless health talks from Baltimore recently, Dr. W. H. Welch referred to "the unavoidable curtailment of available funds" which "places a severe strain upon efficient administration of our city health department," and added these words:

> A very important consideration in these unpredecented days of unemployment is that health and vitality are directly related to means of subsistence and standards of living, especially in the case of infants and children. Every effort should be made to guard against permanent and irremediable damage to health and strength as a legacy of the hard times through which we are passing.

All through the country we find evidence that official health authorities and voluntary organisations are awake to the dangers enunciated by Dr. Welch. An article on Emergency Nutrition by Prof. Henry C. Sherman, of Columbia University, has appeared in the *Child Health Bulletin*, has been given the advantage of partial reprinting in the *Literary Digest*, and with other information of the same kind may now be had from the American Child Health Association (450, Seventh-avenue, New York). Pamphlets have also been prepared by the Bureau of Home Economics (U.S. Department of Agriculture, Washington, D.C.), and will have wide circulation through the county agricultural agents scattered over the vast rural areas of the United States. Add to this publicity the feature stories supplied to newspapers and the special publications of the State health departments, and it is evident that an effective campaign is being waged to prevent malnutrition in the United States.

FERTILITY OF NATIVE WHITE AMERICANS.

Working for the Milbank Memorial Fund's division of research Edgar Sydenstricker,[1] the eminent statistician, writes on the fertility of native white women in a rural area of Western New York. The area includes five townships in Cattaraugus county, with a total population of about 5000, composed almost entirely of native white families of the "older native stock." To 1036 women who had been married only once and had not been permanently separated from their husbands before their forty-fifth birthday, there had been born 2959 live children. The average number of children per wife is thus 2·86. The average of course increases with age from just over one child at age 25 to an average of 3·31 for women aged 45 and over. Yet more than a quarter of the mothers in this group had born either no children at all or only one child. The average, Sydenstricker concludes, is probably sufficient to maintain the size of a relatively healthy rural population. Dividing the rural women into three groups according to the occupation of the husband—farm, labour, or business—and standardising the birth-rate for age, it was found that fertility-rates vary with occupation even in a rural community, the highest rate (2·99) being found in the farm group, and the lowest (1·89) in the business group. The size of the sample for this purpose is small, and the relative fertilities of these groups cannot be accurately gauged by the above figures. But it seems clear that there is an occupational influence. It is of interest too that this occupational difference becomes emphasised with the length of married life, and appears scarcely at all in the first three years of marriage. There is thus no indication that the business group is pressed to use contraceptive methods in early married life by economic considerations. In comparing the fertility-rates in this area for 1929–30 with those of 1910 and 1920 it is found that there has been no significant downward trend in the fertility-rate of farmers of native white stock. Is the general decline of fertility in our "older native stock" a matter not of "lessened vitality" but rather of changing environment? Such are the interesting speculations which this research has aroused.

[1] Milbank Mem. Fund Quart. Bull., January, 1932.

MEDICAL RESEARCH COUNCIL.

ANNUAL REPORT FOR 1930–31.[1]

In presenting the Report for 1930–31 of the Committee of the Privy Council for Medical Research, Mr. Stanley Baldwin, as Lord President of Council, draws special attention to the appointment in July, 1931, of the Agricultural Research Council. Its Charter of Incorporation is closely similar to that of the Medical Research Council, appointed 12 years ago, and coöperation between the two bodies will be assured by the provision that they will have at least one member in common. He mentions the laws of nutritional science and the methods and results by which they can be elucidated, and the study of disease in the animal kingdom, among the foundations on which both agriculture and medicine base their powers.

VITAMIN D.

The Report of the Medical Research Council records fruitful and encouraging results of the year's work. Foremost among these is placed the attainment of accurate knowledge of vitamin D, in terms of its chemical structure and physical properties. The importance of possessing a fixed stable substance by reference to which the vitamin D activity of any foodstuff or remedy can be measured, need hardly be emphasised. A standard preparation of the parent substance, irradiated ergosterol, is already maintained at the National Institute for Medical Research for international reference. The wisdom of

[1] H.M. Stat. Office. Cmd. 4008. 2s. 6d.

the provision made for the redefinition of this international unit in terms of the pure substance, when this should be found, is now apparent; for it seems likely that the isolation of calciferol at the National Institute and the practically simultaneous isolation of the same substance (as vitamin D_2) by Prof. A. Winduas and his colleagues at Copenhagen, will fulfil this condition. The story of great technical difficulties successfully overcome by the team of investigators under the general leadership of Dr. R. B. Bourdillon makes stimulating reading. Practical studies undertaken to explore the possibilities of exploiting for the public benefit the known biological properties of this vitamin are also recorded. The confirmation by a large scale controlled investigation of the view first put forward by Mrs. May Mellanby, on experimental evidence, that the incidence of dental caries in formed teeth might be diminished by the addition of vitamin D to the diet, is described; the results of an investigation into the effect of giving vitamin D to children whose teeth are in the course of development will be published next year, and are expected to bring out the value of the vitamin to an even more striking degree. It is clear that many dietaries regarded as adequate may not really be so.

VIRUS DISEASES.

The chance of finding and identifying the particles of any specific virus by microscopic methods has been greatly increased by the introduction by Mr. W. J. Elford, Ph.D., of a series of graded collodion membranes for use as filters. The "filterable" viruses are so called because they can pass through porcelain and other finely porous filters that hold back ordinary bacteria; the absolute and relative dimensions of the particles of any virus can only be estimated, however, if a graded series of filters is used, with pores not only smaller than those in porcelain filters, but closely uniform in size in each grade. The use of "gradocol" filters indicates, for example, that the virus particles of infective ectromelia, a disease of mice, have a diameter of between 0·1 and 0·15 μ; the virus particles of any of the three types of foot-and-mouth disease are much more minute—being between 0·008 and 0·012 μ; a bacteriophage for the colon bacillus has a diameter of 0·02 to 0·08μ; and the limits of the filtration of the vaccinia virus through graded membranes have been determined by Dr. C. H. Andrewes and Dr. Elford with sufficient accuracy to justify an estimate that its units have a diameter of 0·125 to 0·175μ. Preliminary trials by Mr. J. E. Barnard, F.R.S., give promise of a successful demonstration of the living virus of vaccinia in ultra-violet photomicrographs.

Viruses affecting the nervous system, the virus of herpes, and the viruses of avian tumours are also being studied.

The common cold.—Reference is made to the success with which Dr. A. R. Dochez, of New York, transmitted the common cold to apes by the use of material derived from persons in the early stages of catarrh, and filtered so as to exclude bacteria. Clear evidence was obtained, and subsequently confirmed with human volunteers, that the causal agent belonged to the virus group; the virus could be cultivated outside the body, and after a series of subcultures, still produced typical catarrh in a susceptible individual. Dr. Andrewes visited this work and it is to be repeated in this country with human volunteers from among the students of St. Bartholomew's Hospital. The fact that only a brief immunity succeeds infection by this catarrhal virus unfortunately diminishes the prospect of any rapid attainment of the means of practical control.

Virus studies and cancer—The study of the immunity reactions of the viruses producing malignant tumours in fowls has been pursued by Dr. W. E. Gye and Dr. W. J. Purdy, and correlated with the results of work on the immunity reactions of other known viruses. The evidence obtained supports the view that in these tumour filtrates we are dealing with an infective virus which is similar, as regards the immunity reactions it can elicit, to some of the acknowledged viruses causing specific diseases.

RESULTS OF INSULIN TREATMENT.

An inquiry into the results of insulin treatment at special centres is in progress. One series of over a thousand cases under treatment at one centre in the period 1925–31 is now being submitted to statistical analysis. Almost all the patients received insulin, except those cases in the highest age-group which were of the mild and slowly progressive type which can more conveniently be controlled by diet alone. The figures for young and middle-aged subjects show a great preponderance of cases where good health is maintained against all expectations based on previous experience. Moreover, the real gain is much greater than any figures can show, since these cannot measure the contrast between the full measure of bodily and mental vigour now obtained with the ever-increasing and distressing invalidism which was the lot of the diabetic in pre-insulin days.

THE RELATION OF THE COUNCIL TO MANUFACTURING FIRMS.

The Medical Research Council regard it as one of their primary functions not only to promote the stages of scientific discovery as such within the medical field, but also to facilitate the "availability" of the results of discovery in the interests of the public and the medical profession. Often much time and labour must be given before the yield of a new substance can be brought into the phase of large scale production or a new method be adapted for widespread use. Coöperation between investigation and manufacturing firms has been promoted by the Council in various ways, of which one is the organisation of the Therapeutic Trials Committee.[1] This committee works in close touch with the Chemotherapy Committee, who may be responsible for the experimental work with the drugs or preparations submitted that may be necessary or desirable before their use in man. Consultation with the Association of British Chemical Manufacturers has resulted in the formulation of agreed conditions under which new substances may be submitted for clinical study, and certain clinical trials are already in progress. The Medical Research Council regard the improved machinery for assisting the British production of new therapeutic substances as only an added phase of what has been their normal relationship to manufacturing production for many years. The Council assisted by special research the rapid development in the country of manufacturing insulin, and gave assistance by the study of methods for the production of vitamin D, various organic extracts, and material for inoculation against distemper. On the other hand the chief manufacturing firms have always been ready to offer the results of their experience, the supply of raw materials, or help in other forms. Among the names of firms mentioned in this connexion are the British Drug Houses, Lever Bros., Burroughs

[1] See THE LANCET, 1931, ii., 304.

Wellcome and Co., Bovril, Ltd., Boots Pure Drug Co., and Parke, Davis and Co., to which firms the Council express grateful acknowledgments.

CERTAIN OTHER INVESTIGATIONS.

The effective concentration of *puerperal fever* cases at one centre (Queen Charlotte's Hospital) and the organised study of the disease by clinicians and laboratory workers in intimate association is in progress. The chances of recovery for each patient are said to be much increased by the proper concentration of special clinical experience, combined with appropriate laboratory work. Of the 200 grave cases received in the first 12 months only 30 died, though the usual death-rate of such cases is much higher.

Status lymphaticus.—The collective investigation of "status lymphaticus" organised in conjunction with the Pathological Society of Great Britain and Ireland, has been completed, and the conclusion has been reached that the statistical results obtained provide "no evidence that so-called 'status thymico-lymphaticus' has any existence as a pathological entity."

Reference is made to the completion of the publication of the volumes of "A System of Bacteriology," and to the support offered by the Council to the new periodical *Nutrition Abstracts and Reviews.*

The case for amendment of the British Patent Law, and the history of the attempts made by the Council to bring their views on this subject to the notice of the Government are outlined and a memorandum submitted to the Departmental Committee on the Patents and Design Acts and Practice of the Patent Office is printed in full as an appendix to the Report.

INFECTIOUS DISEASE

IN ENGLAND AND WALES DURING THE WEEK ENDED FEB. 20TH, 1932.

Notifications.—The following cases of infectious disease were notified during the week:—Small-pox, 73 (last week 70); scarlet fever, 1516; diphtheria, 994; enteric fever, 25; pneumonia, 2059; puerperal fever, 38; puerperal pyrexia, 103; cerebro-spinal fever, 52; acute poliomyelitis, 6; encephalitis lethargica, 12; dysentery, 26; ophthalmia neonatorum, 106. No case of cholera, plague, or typhus fever was notified during the week.

The number of cases in the Infectious Hospitals of the London County Council on Feb. 23rd–24th was as follows: Small-pox, 129 under treatment, 10 under observation (last week 118 and 4 respectively); scarlet fever, 1204; diphtheria, 1524; enteric fever, 9; measles, 1337; whooping-cough, 349; puerperal fever, 17 (plus 14 babies); encephalitis lethargica, 220; poliomyelitis, 4; "other diseases," 205. At St. Margaret's Hospital there were 16 babies with ophthalmia neonatorum.

Deaths.—In 117 great towns, including London, there was no death from small-pox, 1 (0) from enteric fever, 65 (21) from measles, 4 (0) from scarlet fever, 67 (11) from whooping-cough, 38 (10) from diphtheria, 57 (15) from diarrhœa and enteritis under two years, and 364 (96) from influenza. The figures in parentheses are those for London itself.

The number of deaths from influenza reported has risen sharply, the fatal cases being distributed fairly evenly over the country. Of the great towns Leicester stands highest with 19 deaths, Sheffield following with 13, Bristol with 11, and Great Yarmouth with 10. The total deaths for the year from influenza so far amount to 2342 against 2083 for the corresponding period of 1931. The fatal case of enteric fever occurred at Watford. Manchester reported 6 deaths from measles, Oldham 5, and West Ham 4. Whooping-cough claimed 13 victims at Birmingham, 7 at Manchester. Liverpool reported 4 deaths from diphtheria.

The number of stillbirths notified during the week was 295 (corresponding to a rate of 42 per 1000 births), including 38 in London.

THE SERVICES

ROYAL NAVAL MEDICAL SERVICE.

Surg. Lt.-Comdr. (D) J. S. F. Horton is placed on the Retd. List with the rank of Surg. Comdr. (D).

The following appts. are notified: Surg. Lt.-Comdr. (D) H. J. Lock to *Rodney;* Surg. Lt. (D) L. F. Fairey to *Sussex;* and Surg. Lt. W. D. M. Sim to *Cambrian.*

ROYAL NAVAL VOLUNTEER RESERVE.

Proby. Surg. Lt. W. Lennon to be Surg. Lt.

ROYAL ARMY MEDICAL CORPS.

Capt. C. Russell to be Maj.

Lt. R. E. Gordon resigns his temp. commn.

M. J. Kohane is granted a temp. commn. in the rank of Lt.

R. D. MacRae to be Lt. (on prob.).

ARMY RESERVE OF OFFICERS.

Lt. L. P. Clarke, R.A.M.C. (T.A.), resigns his commn. in the Res. of Off., and retains the rank of Lt.

TERRITORIAL ARMY.

Col. R. E. Bickerton (Hon. Col. 56th (1st Lond.) Div.), having attained the age limit, retires and retains his rank, with permission to wear the prescribed uniform.

Capt. S. Vatcher resigns his commn.

E. G. Gerstenberg (late Offr. Cadet, Glasgow Univ. Contgt., Sen. Div., O.T.C.) and G. H. Thomson to be Lts.

ROYAL AIR FORCE.

Dental Branch.—The undermentioned relinquish their temporary commissions on return to Army duty: Squadron Leader A. A. McMullan, Major, Army Dental Corps, and Flight Lt. R. E. Edwards, Captain, Army Dental Corps.

DEATHS IN THE SERVICES.

Lt.-Col. John Lawrence Van Geyzel, C.I.E., I.M.S. (retd.), died suddenly at Hotel Beau Lieu, Cannes, on Feb. 20th, aged 74. The son of John Abraham Van Geyzel he was educated at the Royal Academy at Colombo, Ceylon, in which city his father lived, and subsequently he graduated at Aberdeen University M.B., C.M. in 1879. In the following year he entered the Indian Medical Service, and was later appointed District Medical and Sanitary Officer, holding that office till 1887, when he became Chemical Examiner to the Government of Madras and Professor of Chemistry to the Madras Medical College, a post which he held for 24 years. He then became Examiner of Medical Stores to the India Office. Lt.-Col. Van Geyzel was a Fellow of Madras University. He married Catherine Alice, daughter of Mr. W. J. Palmer, of Hertford, by whom he had four daughters.

NEW MIDLAND HOSPITALS.—The two cities of Coventry and Lichfield, so long joined together in one diocese, are simultaneously intending to erect new hospitals. Coventry is about to build an infectious hospital at a cost of £123,500, less £65,000 for which the city council has sold the existing building to the Coventry and Warwickshire Hospital which is in urgent need of more accommodation. The new hospital will have 152 beds with administrative arrangements for an ultimate capacity of 236 beds. At Lichfield the present Victoria Cottage Hospital is to be replaced by a new building on another site at a total cost of £23,500, towards which £13,500 is in hand. There will be 34 beds instead of 21, and five private wards instead of two.

HOSPITAL COSTS.—Stockport Infirmary reports a reduction of about 15*s.* per week in the average cost of an in-patient, in consequence of the fall in the price of commodities; the amount last year was £2 2*s.* 1½*d.* The daily average number of beds occupied was 109, the average stay 17 days, and the average cost of each occupied bed £184 17*s.* 2*d.* At Tamworth Hospital there were 661 in-patients who stayed 22 days on the average, and cost £2 17*s.* 2*d.* per week. Subject to the approval of the Board of Education, Tynemouth education committee has decided to increase the maximum charge to parents for operations on their children for enlarged tonsils and adenoids from £2 2*s.* to £4 4*s.*

OBITUARY

SIR FREDERICK ANDREWES, M.D. Oxf., F.R.C.P. Lond., F.R.S.,

EMERITUS PROFESSOR OF PATHOLOGY, UNIVERSITY OF LONDON.

THE death of Sir Frederick Andrewes, briefly announced last week, took place on Feb. 24th after a short final illness, the sequel to the cerebral attack suffered in New York in 1931, from which he had made so remarkable a recovery. In fact, though he had been slowly losing ground, he had kept a great deal of his regained power and was able, even so late as a month ago, to enjoy a walk and talk in some of the sunnier intervals of a London winter.

Prof. Andrewes was in the seventy-third year of his age and, as he told the Abernethian Society in the delightful reminiscences communicated at the time of his retirement from St. Bartholomew's, he was born about the time at which bacteriology was taking its first tentative steps. It was, however, many years before he was drawn to the study of that branch of science. Always an eager student of nature he passed from school to Christ Church, Oxford, as a Junior Student, and following his scientific bent took first-class honours in the school of natural science. A year later he was awarded the Burdett-Coutts scholarship for the study of geology and of natural science as bearing upon geology. Already he had begun his medical career by gaining an open scholarship at the medical school of St. Bartholomew's; in 1886 he was elected to a medical fellowship at Pembroke College, and in 1887 proceeded to the medical degree of his University. In his student days he made his first essay in bacteriology by staining and proudly exhibiting the bacillus of Koch, at that time an entirely novel procedure in clinical medicine. It was not however till 1890 that the influence of Klein, the real Father of Bacteriology in this country, turned his serious attention to this subject. It was in Klein's laboratory, in company with a small band of enthusiasts for the new science, that Andrewes began what were to be the determining studies for his future career. In the next seven years, although he continued his bacteriological work, it appeared likely that his life would be devoted to clinical medicine. He had served the usual junior appointments at his hospital, had been casualty physician, and had been elected assistant physician to the Royal Free Hospital. In these posts he had established a reputation for sound clinical work and able teaching; his ideas were then wholly of clinical practice and of a place on the staff. Yet when in 1897 his intimate friend and forerunner, Kanthack, the first lecturer on pathology at St. Bart.'s, migrated to Cambridge to become professor of pathology, Andrewes seemed to be his obvious successor. He hesitated for a little, as was natural, but (to quote his own words) realised that he loved laboratory work more than out-patients—and accepted the task. So it was at the comparatively mature age of 38 that he entered upon the work which engrossed his activities for the next 32 years. How successful that work proved is common knowledge to all St. Bart.'s men, but his reputation grew quickly outside the walls of his own school, and he soon became recognised as one of the builders of the pathological and bacteriological science which now forms so essential a partof the foundations of medicine and surgery. Rapid and striking as the change has been in the period since he was thus appointed, he still looked forward to wider developments, and quite recently in conversation sketched the future of pathological science, ever enlarging its range till it should embrace the whole activities of medical work.

SIR FREDERICK ANDREWES.

[Photograph by J. Russell.

The department at St. Bart.'s to which Andrewes was appointed in 1897 at first consisted only of himself and Dr. J H. Drysdale and occupied two rooms, but in a few years it grew to comprise many other workers and assistants. His share in this development was of course part of the general rise in the importance of pathology, yet in a particular sense he did more than any other pathologist to direct growth of the new science for the somewhat devious route which he had followed to attain to a leading position in pathology had given him a wider outlook on the relationship between pathology and medical science than was, or even now is, at all common. At his own school his interest in clinical and sanitary work was maintained by his appointments as physician to the nursing staff and sanitary officer to the hospital, and it was these intermissions from the laboratory bench, among other influences, which gave him the experience and vision which enabled him to link up the advancing science with the older departments of medicine. Bacteriology naturally claimed much of his attention, but for many years he spent time in the pathological museum of the hospital, revising and adding to that collection and steadily increasing his own knowledge till he had become an acknowledged authority, not only in naked-eye but in microscopic pathology. One of his earlier papers on the histology of Hodgkin's disease, at any rate in this country entirely original work, has stood the test of time. Meanwhile his reputation was spreading in wider circles, and he was one of the first men to be appointed to the committee which later became the Medical Research Council.

It was on this committee and on its various sub-committees that he did what was perhaps his most valuable work, especially during the period of the war. The part he played in the organisation and mobilisation of the forces required to deal with the new medical problems which crowded in during those four years is probably known to few; there were in fact not many of the urgent pathological investigations to which his knowledge, his wide experience of men and methods, and his balanced judgment of results did not materially contribute. This work was recognised in 1920 by the award of a knighthood. Other honours came to him. He was elected a Fellow of the Royal Society, an Honorary D.C.L. of Durham University, Croonian lecturer of the Royal College of Physicians of London in 1910, and Harveian orator in 1920.

Perhaps the work which was his most favoured field was the study of the streptococci. Alone and with others he wrote much on this subject, and though later work, some of it his own, has invalidated many of the actual results, the labour devoted to these studies served to erect much of the essential scaffolding of bacteriological theory. Of recent years he had returned to these aspects of bacteriology and had specially studied the antigenic structure of bacteria as demonstrated by their agglutinative reactions, amassing knowledge regarding the variation of bacterial strains. This work he had continued after retirement from his professorship, and at the time of his death he was occupied with the revision and collation of his research work. Apart from his main contributions to pathology he had on various occasions charmed the more intimate circle of his friends by "parerga" which none but he could have written. One of these entitled "The Romance of the Streptococci" was a skit on his own lectures; another his own account of recovery, at first slow then rapid, of his power of speech after the cerebral attack. Both of these have the vivid presentation, the perfected phrase, the humorous innuendo, and the balanced style which always marked his papers.

Andrewes was not, as measured by the standard of the past, a great pathologist; he would himself have claimed only to have cultivated his small patch of garden to the best of his ability, with the same care and eager pleasure with which he occupied his leisure in his rock-garden. But he was certainly in this country one of the leaders in the development of bacteriological and pathological science; a builder with power to select and reject material and results so as to form firm foundations. His influence has been and will be more widely felt in the example he gave of tireless industry, scrupulous accuracy, skilful exposition, critical judgments, and a power of inspiring all who came in contact with him.

Sir Frederick Andrewes married in 1895 Phyllis Mary, daughter of John Hamer, J.P. Their only son is on the scientific staff of the National Institute of Medical Research.

CHARLES WALKER CATHCART, C.B.E., M.B. Edin., F.R.C.S. Eng.

By the death of Mr. C. W. Cathcart the Edinburgh school loses a distinguished veteran teacher. It is true that Cathcart belongs rather to the last generation than to this; but in his eightieth year he was still a living presence in both the medical and the social life of Edinburgh.

Graduating as M.A. in 1873, and as M.B., C.M. in 1878, he attained the surgical Fellowships of England in 1879 and of Edinburgh in 1880, having previously served as house surgeon to Annandale at the Royal Infirmary. In due course he joined the staff of this hospital as assistant surgeon, and, tying for the Liston Victoria jubilee prize of the Royal College of Surgeons of Edinburgh in 1893, became full surgeon in 1899. He was an authority on surgical pathology, and for a long time was conservator of the College of Surgeons museum. He conducted classes on systematic and operative surgery in the Edinburgh School of Medicine and later was a lecturer in the Royal Infirmary. At the outbreak of war, in 1914, he held the rank of lieut.-colonel in the Territorials, and he had a great deal to do with the equipping of the Second Scottish General Hospital at Craigleith. Afterwards he was surgeon to the general military hospital at Bangour, and at the end of the war he was appointed to the staff of the Edenhall Hostel for Limbless Soldiers and Sailors. For his war services he was created C.B.E.

These are the externals of Cathcart's career as a surgeon. But such a record gives little idea of his personality. "He was a man of great originality," writes a colleague; "one could not talk with him for five minutes without hearing some fresh idea. In the laboratory he was as facile as in the surgical ward. Cathcart's microtome was familiar to medical students more than 40 years ago. He was continually devising something new. He was a born teacher, which means that he was always a student and understood the student mind from within. The 'Surgical Handbook' of Caird and Cathcart was the constant companion of the house surgeon. Both men had ingenious minds, and their combination as authors would be hard to better. As late as 1928, Cathcart, in collaboration with Mr. Hartley, produced a text-book with the title 'Requisites and Methods in Surgery.' Early in the war he was, I believe, the first to suggest sphagnum moss as a convenient surgical dressing, and started the organisation for collecting it. In social affairs he was continually working to improve the life of the poor—in diet and otherwise. But these high-tension activities had to be paid for and, although an athletic man in his early days, he was forced in middle life to take a year's sea voyage. It was not quite successful in clearing off his insomnia, and he found the voyage, which included Australia, less a rest than a discipline. But Cathcart, on resuming work at home, became once more the same fertile, quiet influence in the medical school, and it was a stimulus to hear him at the Medico-Chirurgical Society. In the war his only son was killed, and the death was a great blow to a man of highly emotional nature; but as a memorial to the son he devised a play centre in one of the poorer quarters. He was, in fact, a practical sociologist as well as a surgeon.

"In the story of the Medical School of Edinburgh Cathcart will be found as a man in the great succession of Edinburgh surgeons; modest and unpretentious, yet creating an atmosphere of conscientious devotion to duty and establishing a record of technical work on the highest practical plane."

WILLIAM DAVID DYE, D.Sc., F.R.S.

We regret to record the death of Mr. William D. Dye, D.Sc., A.C.G.I., at the early age of 44. Dr. Dye was the third son of Mr. Charles Dye, J.P., of Portsmouth. After taking the D.Sc. Lond., and

(*Continued at foot of opposite page.*)

CORRESPONDENCE

TUBERCULOSIS OF CHILDHOOD.

To the Editor of THE LANCET.

SIR,—In view of the report recently issued by the Medical Research Council (P. D'Arcy Hart: The Value of Tuberculin Tests in Man) we feel that Dr. Ernest Fletcher's paper in your last issue was somewhat provocative in nature. While allowing for his attempt to meet expected criticism there are, surely, many who would challenge the manner in which he has selected his "normal" children. Many of the findings at the Henry Phipps Institute (in spite of vast expenditure of time, money, and energy) have not been confirmed by workers in this country. Dr. Fletcher is to be commended on his deviation from Opie and McPhedran's routine. Radiology however is a good servant but a bad master, and we feel that some at any rate of Dr. Fletcher's diagnoses, based almost solely on radiological findings, are open to serious criticism.

One of Hart's main conclusions is that a child of school age, who fails to react to tuberculin, is not suffering with tuberculosis, whatever else may be the cause of his signs or symptoms. The illustrations of two of the cases, particularly in view of the remarkably short after-histories, may well be those of incompletely resolved pneumonic conditions. Such cases are far from rare in tuberculosis dispensaries with adequate X ray facilities. Nor does Dr. Fletcher state on what he bases his diagnosis of "tuberculous tracheo-bronchial glands" in four children, in only two of whom was a positive tuberculin test recorded. If this again was founded only on radiological evidence we may be forgiven for quoting the conclusions reached by the committee appointed by the National Tuberculosis Association of America to study the "Clinical and X ray findings in the chests of normal children":

"*The normal chest.*—The normal chest of the child from the roentgenological standpoint is subject to such wide variations within normal limits as to be beyond the possibility of exact description.

"*Hilum shadow.*—The conglomerate shadow, commonly called the hilum shadow, when found lying entirely within the inner third or zone of the lung area can be disregarded (or regarded as normal), except where it is made up of a solid mass of homogeneous shadow giving undoubted evidence that it represents a growth or mediastinal pleurisy.

"*Calcified nodes.*—Calcified nodes at the root of the lung, without evidence of lung disease, are of no significance except as a possible evidence of some healed inflammatory condition, possibly but not necessarily tuberculous. They are a common finding in normal chests."

Wessler and Jaches, in remarking on enlarged root shadows, also state that "to regard these shadows as evidence of involvement of the lung root in a tuberculous infiltration is fatal to a rational interpretation of the plates; a large percentage of mankind would then be pronounced clinically tuberculous and the Rontgen diagnosis would be reduced to an absurdity."

Dr. Fletcher reaches the conclusion that tuberculosis of childhood is liable to develop without close contact with a known case. He does not state whether he has taken the necessary step of securing the examination of the parents of these children. In a preliminary series investigated in the Bermondsey Tuberculosis Dispensary, in those families where it was possible to secure a thorough examination of both parents, we found that of 16 infants who had died of miliary or meningeal tuberculosis 15 had been in close contact with definite cases of pulmonary tuberculosis. In eight of these cases the father was suffering with pulmonary tuberculosis, and in four cases the mother, and in the remaining three instances a known (sputum positive) case was living in the same house. Further, in a series of ten consecutive cases of pulmonary tuberculosis (sputum positive) in children of school age, eight of them had been in recent contact with definite cases of the disease. To quote Dr. Fletcher, "it seems that there is still a large amount of accidental tuberculosis abroad," but in England, to judge from our experience, infection in childhood is not quite so accidental!

We have no desire to enlarge on the disadvantage of labelling a child as tuberculous without justification, but this question is of more than academic interest. The mere notification of a case of tuberculosis (and this is compulsory) sets in motion administrative action which, though it might vary from one area to another, involves the expenditure of money by the State. Clerks are employed to keep registers. Returns, often in duplicate, must be made to higher authorities; nurses are employed to visit the homes; tuberculosis officers are employed to supervise this work, to examine the contacts, and to effect prompt treatment when necessary. One unjustifiable notification, therefore, leads to expense by the State, even if nothing further is done. If, however, institutional treatment follows, as it often does quite automatically, the expense mounts at once.

At a time when the country is faced with a severe economic crisis surely these factors should be considered very closely. Dr. Burrell has repeatedly stressed the inadvisability of expending on the treatment of debilitated children large sums of money which have really been earmarked for dealing with true cases of tuberculosis and which would probably lead to greater efficiency if applied solely for this purpose. A glance at the reports of the Lancashire tuberculosis scheme will reveal that adequate provision can be made for all tuberculous children at relatively small cost, leaving the administration a freer hand to deal with the major problems confronting it.

We are, Sir, yours faithfully,

C. H. C. TOUSSAINT,
E. J. MACINTYRE.

Public Health Department, Tuberculosis Dispensary, Grange-road, Bermondsey, S.E., March 1st, 1932.

(*Continued from previous page.*)

getting experience at the B.T.H. works, Rugby, Dr. Dye was appointed a member of the staff of the electrical standards division of the National Physical Laboratory, becoming head of this department on the transference of his chief Mr. F. E. Smith (now Sir Frank Smith) to the Admiralty. Dr. Dye was chiefly interested in the methods for evaluating and standardising the primary electrical units and did much highly accurate work upon inductance and capacity. He also contributed to the development of modern methods of measurement of time and this brought him into contact with the Radio Research Board and for several years he acted as chairman of the Commission on Radio Standards. Dr. Dye was elected a Fellow of the Royal Society in 1928, and his early death will be felt in more than one branch of scientific work.

ARTIFICIAL PNEUMOTHORAX.

To the Editor of THE LANCET.

SIR,—The note in your issue of Feb. 27th (p. 450) by Dr. J. D. Macfie and Dr. A. J. P. Alexander on the follow-up of artificial pneumothorax cases is of great interest and importance. With their assessment of the practical value of this method of treatment I am entirely in agreement, but it would be of special interest to know further details of the cases that have died, particularly as regards the mode of death. It is to be hoped that they will publish a more extended study at a later date.

It is, I think, to be regretted that more investigation has not been carried out in this country on a really large scale as to the ultimate cause of death in cases of pulmonary tuberculosis treated by artificial pneumothorax, with special reference to the spread of disease in the contralateral lung. Even now, despite all the work that has been done by different observers and the vast amount of material available for statistical purposes, we are still to a large extent ignorant of the factors which should go to form the criterion of suitability in our selection of cases for pneumothorax treatment. My impression, after some years of clinical experience, is that phthisical patients fall roughly into three groups: those that go downhill whatever treatment is employed, those that recover and achieve permanent arrest in almost any circumstances, and those that occupy a middle position, so to speak, in whom one's treatment may well be the deciding force between life and death. I am inclined to think that it is the last group which furnishes the most successful results in pneumothorax treatment. Moreover, there is, I believe, a strong impression among many who have worked at the subject that while the immediate prognosis of pulmonary tuberculosis is more favourable in A.P. cases, the ultimate outlook—i.e., as regards length of life—is better in patients in whom cure has been attempted by more natural means.

I am, Sir, yours faithfully,

MAURICE DAVIDSON.

Wimpole-street, W., Feb. 28th, 1932.

A CAMP FOR UNEMPLOYED MEN.

To the Editor of THE LANCET.

SIR,—We believe that it is within the experience of all medical men in general practice that chronic unemployment almost inevitably leads to deterioration of mental and physical health, and that this adds to the difficulty of re-entering organised industry. With unemployment as widespread as it is now, the progressive inefficiency in the community from this cause becomes a menace not only to the happiness of millions, but to our communal ability to take the initiative if opportunities present themselves for a revival of trade, and to our capacity for reorganising our social structure if progressive rationalisation of industry demands it.

We are closely associated with a group of social workers known as the Order of Wooderaft Chivalry, which has set on foot a scheme of national non-military service. This has been devised so as to give opportunity to all young men who have either voluntary or involuntary leisure, to devote it to a full-time camp course of training, directed to the maintenance of physical fitness, mental alertness, initiative, and citizenship.

We are not yet ready to undertake the regeneration of demoralised men, though we hope to be so soon. Our first pioneer group, which will go into camp in March, must grow at first by the addition of men of courage, initiative, and vision, capable of building up a tradition of hardihood and mutual help; and the purpose of this letter is to ask that the names and addresses of such men—great numbers of whom must be known to your readers—may be sent to the hon. secretary at 2, Devonshire-place, W.1. They should be between the ages of 18 and 25. If they are entitled to unemployment or transitional benefits, the Ministry of Labour has agreed that these may be paid over for their maintenace while under training.

We shall be very glad to supply full information to anyone interested.

We are, Sir, yours faithfully,

(Sgd.) I. NORMAN GLAISTER, D. L. TUCKER, E. SCHOFIELD, R. TURNER, B. STANLEY, A. WESTLAKE. F. TALBOT,

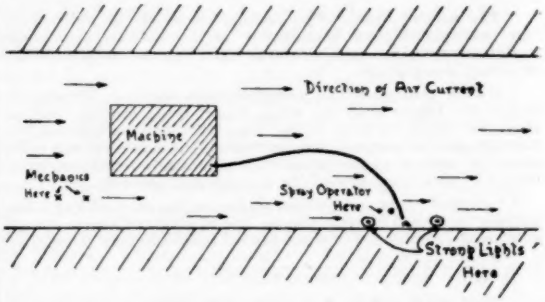
2, Devonshire-place, W., Feb. 27th, 1932.

ASBESTOS IN LONDON TUBE RAILWAYS.

To the Editor of THE LANCET.

SIR,—With reference to the annotation on page 410 of your issue of Feb. 20th, we, as inventors of the process referred to, give you herewith a few particulars, which we trust will enable you to arrive at a better conception of the work which is at present being carried out in the London Underground tunnels. Particular attention is drawn by you to the Home Office inquiry into the effects of asbestos dust on the lungs, and it will be seen that ventilation and the replacement, wherever possible, of dry processes by wet, form the basis of the safeguards recommended, of which advantage has been taken by the designers of our apparatus.

As will be seen on examination of "Methods for Suppressing Dust in Asbestos Textile Factories," issued by



the Home Office (1931), a system of tunnels shown on pages 11, 31, and plate opposite page 36, which are reproductions of photographs taken in our factory, provides an incoming supply of fresh air, removing any possible traces of dust from the vicinity of the worker, and we would point out that this is exactly what takes place in the Underground tunnel, the apparatus being so arranged that the operator is kept constantly supplied with fresh air, and any dust or atomised adhesive is blown away from him immediately by the natural current of air flowing through the tunnel, assisted where necessary by mechanical means, for which full provision has been made.

In addition, a special study has been made of the question of suitable adhesives, so that sufficient liquid can be used through the second projector to completely saturate the fibre delivered from the other

nozzle. This in itself removes the possibility of dry particles of fibre escaping into the surrounding atmosphere. We would add that, contrary to your assumption, a minimum air pressure is maintained at the fibre nozzle, and perhaps the rough sketch attached will enable you to see how full advantage has been taken of the natural conditions prevailing in the tunnel system.

Before this work was commenced, a very thorough investigation of our process was made by the engineers of the London Underground railways, and we can assure you that no pains are being spared to make this thoroughly satisfactory in every respect.

We are, Sir, yours faithfully,

J. W. ROBERTS, LTD.,
Armley, Leeds, Feb. 29th, 1932. Asbestos Manufacturers.

BLOOD GROUPING OF MONGOLIAN IMBECILES.

To the Editor of THE LANCET.

SIR,—I am grateful to Mr. Geoffrey Keynes for raising a point which perhaps was not made clear in my paper of Feb. 20th. It was certainly not my intention to assume the existence of a linkage between Mongol characters and either agglutinogen. My argument, such as it was, relied entirely on the fact that A and B are inherited as dominant characters, so that, for example, a high incidence of one of these characters among ancestors tends to produce a high incidence of this same character among the descendants. The distribution of the blood groups in the "Mongolian imbeciles," therefore, shows that among their ancestors persons belonging to populations relatively rich in B (e.g., Eastern races) have not been more frequent than among the ancestors of the individuals in the control group.

Such a conclusion, I stated, seemed inconsistent with Dr. Crookshank's views. However, in his letter, Dr. Crookshank says that my observations do not conflict with his views, and of this he is the best judge. I am sorry to have misrepresented him.

I am, Sir, yours faithfully,

Colchester, Feb. 29th, 1932. L. S. PENROSE.

CORRECTION.—In our issue of Nov. 28th, writing from Semaphore, Australia, in reference to Darwin's naval service, Dr. F. Lucas Benham gave the name of the Captain of H.M.S. *Rattlesnake* as Owen Smyth. He should have written Owen Stanley, he informs us, a name whose familiarity is due to the fact that it has been bestowed on the highest mountain in British New Guinea.

PARLIAMENTARY INTELLIGENCE

NOTES ON CURRENT TOPICS.

DANGEROUS DRUGS—IMPORT DUTIES.

ON Feb. 25th the Dangerous Drugs Bill was agreed to on Report.

The Committee stage being completed, the Import Duties Bill was reported to the House on Feb. 23rd, after which amendments were accepted (1) providing that scientific films should continue to enjoy the privilege conferred on them by the Act of 1928, and (2) putting cinchona bark, cork, cork shavings, and waste cork on the free list. On Feb. 25th the Bill was read the third time by 442 votes to 62—majority 380.

In the House of Lords on Feb. 29th the Import Duties Bill passed through all its stages and was given the Royal Assent.

VIVISECTION AND CHEMICAL WARFARE.

On March 1st, in the House of Commons, Mr. GROVES presented a petition against the legal practice of vivisection and the use of poison gas in warfare. The petition was signed by over 160,000 citizens.

SCIENTIFIC SLAUGHTER OF ANIMALS.

On March 1st, in the House of Commons, Lieut.-Colonel MOORE asked leave to present a Bill to provide for the humane and scientific slaughter of animals. He said that he introduced a similar Bill last year, but after it had passed half-way through the committee stage the national crisis supervened. The Bill sought to ensure that all animals killed for human food should be slaughtered by a mechanical instrument, instead of by the present pole-axe method. Of 1500 local authorities, 500 had voluntarily adopted the by-law of the Ministry of Health to this end, and many practising butchers had followed their example. The Bill was promoted by the Royal Society for the Prevention of Cruelty to Animals. He had decided, to avoid criticism, to withdraw pigs from the scope of the Bill. Sixteen million animals were involved yearly, and because of the voluntary action of certain local authorities 4,000,000 of them had been relieved of suffering.

Leave was given to introduce the Bill, and it was brought in and read a first time.

HOUSE OF COMMONS.

WEDNESDAY, FEB. 24TH.

Establishment of Naval Surgeons.

Mr. RALPH BEAUMONT asked the First Lord of the Admiralty whether the establishment of naval surgeons was complete; and, if not, what was the deficiency.—Lord STANLEY (Parliamentary Secretary to the Admiralty) replied: The establishment of naval medical officers is not complete, the deficiency at present being approximately 60.

Mr. BEAUMONT: Is it proposed to take any steps to make up that deficiency?

Lord STANLEY: Yes, the whole question of the conditions of service, recruitment, &c., in the medical branches of the three Services is under consideration by an inter-departmental committee.

Prison Administration.

Mr. GORDON MACDONALD asked the Home Secretary what action he intended to take with regard to the request made to him for the setting up of a Royal Commission to investigate and report upon the whole question of prison administration with special reference to prison industries.—Sir HERBERT SAMUEL replied: I am of opinion that the system of prison industries needs review. As I stated recently in answer to a question, a committee was appointed by my predecessor to consider the best methods of dealing with persistent offenders, and I understand that the report of that committee, which is expected to be completed in a few weeks from now, will call attention to the need for the improvement of prison industries in connexion with other proposals. When this report is received, I will consider what would be the right procedure to adopt, but, as at present advised, I do not think that the appointment of a Royal Commission would be the best means of investigating the somewhat detailed and technical questions that are involved, or would conduce to the speedy action which may prove to be desirable.

Encephalitis and Vaccination.

Mr. GROVES asked the Minister of Health how many cases of encephalitis following vaccination had been reported either direct to the Ministry of Health or through the Andrewes and Rolleston vaccination committees, from the first sitting of the Andrewes committee down to the present time; how many of these had been included in the cases admitted to be cases of injury to the nervous system resulting from vaccination; and how many fatal cases were similarly reported.—Sir E. HILTON YOUNG replied: During the period under consideration by the Andrewes Committee and the Rolleston Committee—i.e., since November, 1922—202 cases of encephalitis following vaccination have come to the notice of my department. Of these cases, 197 were,

as the result of inquiry, regarded as cases of post-vaccinal encephalitis, and 102 proved fatal. In the remaining five cases, of which two died, there appeared to be no reason to associate the encephalitis with the antecedent vaccination.

THURSDAY, FEB. 25TH.

Mental Deficients and Paternity Cases.

Sir JOHN HASLAM asked the Home Secretary whether his attention had been drawn to a recent paternity case tried at the Bolton county police-court, wherein a man who acknowledged his responsibility escaped his liability, because the justices had to dismiss the application owing to the claimant being of too weak an intellect to understand the nature of an oath; and whether he would introduce, at an early date, legislation to remedy that state of things.—Sir HERBERT SAMUEL replied: This case is most exceptional and probably unique. The complainant appears to have been of insufficient capacity to understand the nature of an oath or of the proceedings, and it would cause injustice if an order could be made against a man denying paternity without the evidence of the mother. In this case the man is reported to have been willing to admit paternity and, if so, the right course to pursue would be to invite him to enter into an enforceable agreement for the payment of maintenance to the mother.

Medical Benefit Regulations.

DR. HILLMAN asked the Minister of Health whether, seeing that he had decided not to proceed with the proposed redraft of parts of the medical benefit regulations dealing with fee-charging, and that the Insurance Acts Committee of the British Medical Association acquiesced in this decision, he would take the opportunity of making it clear that there was no connexion between the proposed redraft and the deduction from insurance doctors' remuneration made as an emergency economy last October.—Sir E. HILTON YOUNG replied: I am glad to have an opportunity of repeating what I indicated in reply to a question on this subject by my hon. friend on Dec. 3rd, that the proposal to redraft the relevant parts of the regulations was initiated by the department in September, 1930, and was therefore entirely unconnected with the proposals for securing emergency economies in the cost of medical benefit which became necessary a year later.

Mr. RHYS DAVIES: Will the right hon. gentleman tell the House why the proposed draft has been dropped?

Sir E. HILTON YOUNG: It was dropped because it was considered not to be necessary.

Sanatorium Treatment of Tuberculosis.

Mr. DAVID GRENFELL asked the Minister of Health if he would give the most recent figures showing the result of treatment of tuberculous patients at sanatoriums in the United Kingdom, with the number of patients admitted, the number who were passed out as cured, and the number where treatment was unsuccessful.—Sir E. HILTON YOUNG replied: The following are the figures in respect of England and Wales: The number of persons admitted for the treatment of tuberculosis to approved residential institutions in England and Wales in the year 1930 was 44,517. The number of persons discharged in the same year from such institutions with their disease quiescent was 9410. The number who died in the institutions in the same year was 5073. A person who has suffered from tuberculosis is not considered to be cured of the disease until it has been quiescent for five years if it is pulmonary, or for three years if it is non-pulmonary. It is not therefore usually possible to say at the date of discharge from an institution whether the treatment has been permanently successful.

Milk and Butter-fat Standard.

Mr. GLOSSOP asked the Minister of Health whether in view of the fact that under the existing law milk might be sold containing less than 3 per cent. butter-fat, Government standard, provided that the farmer could prove that such milk was as it came from the cow, he would consider the revision of the law in this matter.

Brigadier-General CLIFTON BROWN asked the Minister of Health whether, seeing that the law did not require that milk shall contain any particular percentage of fat, and that it was not an offence to sell milk containing less than 3 per cent. of fat, he would alter the sale of milk regulations which lay down that milk containing less than 3 per cent. of fat was presumed to be adulterated until the contrary was proved; and whether he would put the onus of proof on the prosecutor as was the case in other similar regulations.—Sir E. HILTON YOUNG replied: I have received representations with regard to the amendment of the law in the directions suggested, and will consider the matter in consultation with my right hon. friend, the Minister of Agriculture and Fisheries.

Labelling of Imported Meat.

Mr. HURD asked the Minister of Agriculture what means were taken to lead local authorities to enforce the distinct labelling of imported meat in the shops, as required by the Sale of Food Order, 1921; and whether he was aware of the laxity of some authorities in this matter.—Sir JOHN GILMOUR replied: As my hon. friend is no doubt aware, the Sale of Food Order of 1921 has now been revoked with the exception of the provisions relating to meat. This portion of the Order has been continued in force from year to year under the Expiring Laws Continuance Act. Power to enforce the provisions lies with the local authorities, and I have no power to compel them in the matter.

TUESDAY, MARCH 1ST.

Poison Gas Experiments on Animals.

Mr. GROVES asked the Financial Secretary to the War Office the reason why no reports had been issued on the work of the chemical warfare medical committee since 1920 on the question of poison gas experiments on animals; and where accounts of the experiments performed during the last 11 years and their results might be obtained.—Mr. DUFF COOPER (Financial Secretary to the War Office) replied: This committee was dissolved in 1919 and research on such problems has been continued by the Chemical Defence Committee. Information as to the number of experiments performed is given in the Home Office returns of experiments on living animals of which the last issue was dated July 29th, 1931.

Blindness and Insanity Due to War Service.

Mr. MCGOVERN asked the Secretary of State for War the number of persons who were blinded and driven insane as a result of service in the late war, and the numbers who lost either an arm or a leg or both arms or both legs.—Major TRYON (Minister of Pensions) replied: The approximate numbers of pensions in the categories mentioned at the end of 1931 were as follows: Cases of total blindness, 975; of mental disorder in mental hospitals, 6300; and cases of amputation of a leg or arm, or of both arms or both legs, 34,500.

Appointments

Certifying Surgeons under the Factory and Workshop Acts: ARNOTT, D., M.B., Ch.B. Edin. (Shaftesbury); SEARS, E. H., L.R.C.P. Lond., M.R.C.S. (Lyndhurst); and WORSLEY, A. (Malvern).

Births, Marriages, and Deaths

BIRTHS.

HUBBLE.—On Feb. 24th, the wife of Douglas Hubble, M.B., of Kedleston-road, Derby, of a daughter.

MCCURRICH.—On Feb. 23rd, at Palmeira-avenue, Hove, the wife of H. J. McCurrich, M.S., F.R.C.S., of a son (stillborn).

SWINDELL.—On Feb. 25th, at Westwood-road, Southampton, the wife of Dr. R. S. Swindell, of a daughter.

DEATHS.

ANDREWES.—On Feb. 24th, at Windy Gap, Merton-lane, Highgate, Sir Frederick William Andrewes, M.D., F.R.S., in his 73rd year.

APPERLY.—On Feb. 24th, suddenly of pneumonia, at Chasemere, Kingswood, Surrey, Herbert Apperly, L.D.S. R.C.S. Eng.

CATHCART.—On Feb. 22nd, at a nursing home, Edinburgh, Charles W. Cathcart, F.R.C.S. Eng. and Edin., of Newbattle-terrace, Edinburgh.

GARDNER.—On Feb. 27th, at Imberway, Warminster, William Gardner, M.R.C.S., L.R.C.P., in his 75th year.

VAN GEYZEL.—On Feb. 20th, suddenly, at Hotel Beau Lieu, Cannes, John Lawrence Van Geyzel, C.I.E., Colonel I.M.S. (retired), aged 74.

N.B.—A fee of 7s. 6d. is charged for the insertion of Notices of Births, Marriages, and Deaths.

QUEEN'S INSTITUTE OF DISTRICT NURSING.—Princess Alice, Countess of Athlone, last week presented long service badges to "Queen's" superintendents and nurses who have completed 21 years' service. Since the last meeting of the council 32 associations have been affiliated and the names of 405 nurses have been placed on the roll of Queen's nurses. At the end of last year there was a deficit, and had it not been for the large sum raised by the gardens scheme "the institution would have been faced with a very serious financial crisis."

MEDICAL NEWS

Society of Apothecaries of London.

At recent examinations the following candidates were successful :—

Surgery.—A. Kobylivker and W. W. Parry, Guy's Hosp.

Medicine.—R. R. Clipstein, St. Mary's Hosp.; B. Dayal, Univ. of Durham; W. Hertzog, Guy's Hosp.; J. L. F. King, Charing Cross Hosp.; M. S. Mules, Royal Free Hosp.; C. E. Nicholson, Charing Cross Hosp.; G. W. Sturgess, St. Bart.'s Hosp.; F. A. Trowbridge and C. F. Vieyra, St. Mary's Hosp.; and L. G. Yendoll, Guy's Hosp.

Forensic Medicine.—G. A. Dingemans and W. S. Pitt Payne, Guy's Hosp.; and L. R. C. Rose, Univ. of Manchester.

Midwifery.—G. W. Aston, Univ. of Birmingham; L. A. De Dombal, Univ. of Sheffield; and W. Hertzog and A. Kobylivker, Guy's Hosp.

The following candidates, having completed the final examination, have been granted the Diploma of the Society entitling them to practise medicine, surgery, and midwifery: R. R. Clipstein, W. W. Parry, W. Hertzog, G. W. Sturgess, J. L. F. King, and C. F. Vieyra.

Royal Society of Arts.

Mr. J. C. Drummond, D.Sc., professor of biochemistry, in the University of London, will give three lectures, on Recent Researches on the Nature and Function of Vitamins, at the house of the Royal Society of Arts, John-street, Adelphi, London, W.C., on Mondays, April 18th and 25th, and May 2nd at 8 P.M.

Congress on Biliary Lithiasis.

An International Congress on Biliary Lithiasis will be held at Vichy from Sept. 19th to 22nd. The principal subjects of discussion will be the Sequelæ of Cholecystectomy, the Medical and Hydro-mineral Treatment of Cholecystitis in Biliary Lithiasis, and the Cholelithiatic Liver. Inquiries should be addressed to the secretary-general of the Congress, 24, Boulevard des Capucines, Paris.

Mental Deficiency and Allied Conditions.

A course of lectures for medical practitioners on Mental Deficiency and other Mental Conditions Closely Allied to it, will be held at the University of London, Imperial Institute-road, South Kensington, London, S.W., from April 11th to 23rd, supplemented by a course of clinical instruction. The lecturers will include Dr. A. F. Tredgold, Dr. F. C. Shrubsall, Prof. Cyril Burt, Dr. W. Rees Thomas, Dr. William Moodie, and Dr. Emanuel Miller. Further particulars may be had from Miss Evelyn Fox, c/o University Extension Dept., at the above address.

Alfred Fripp Memorial Fellowship.

On Feb. 26th, at University College, Mr. Stanley Baldwin delivered the first of the 20 annual lectures on Happiness and Success, for which Sir Alfred Fripp left a legacy. After the lecture Lord Goschen, the chairman, handed over to the treasurer of Guy's Hospital the trust deeds of the Alfred Fripp Fellowship, which has been founded to promote research in the psychology of children. Election to the Fellowship will be by a board of electors consisting of the physician for diseases of children, the superintendent and the dean of the medical school of Guy's Hospital as ex-officio Members, and of five Members coöpted by them. The Fripp Fellow will be allowed opportunities of study in the children's department of the hospital, and candidates must be qualified men or women, unmarried, and not more than 30 years of age. The stipend will be £300 per annum, and the Fellowship is to be regarded as a whole-time appointment, though it may be held concurrently with a demonstratorship. It will be held ordinarily for two years.

Bacteriological Investigation of Puerperal Sepsis.

By arrangement between the Ministry of Health and the Medical Research Council, Dr. Dora Colebrook, has been appointed by the Medical Research Council to work in the new laboratories of Queen Charlotte's Hospital, Hammersmith, where a number of agglutinating sera are available for typing puerperal strains of streptococci. Her appointment gives effect to a recommendation of the Departmental Committee on Maternal Mortality, that combined epidemiological and bacteriological investigation should be more often undertaken, with a view to defining the responsibility of the throat "carrier" and to investigating the rôle played by other infective conditions in persons in contact with the patient. Dr. Colebrook's time will be fully occupied with special typing. Medical officers of health are invited to communicate directly with Dr. Colebrook in the event of an outbreak and to send swabs from the cervix uteri or upper vagina, and from tonsils and nose of the patient and the contacts. By this means it is hoped to determine the bacteriological nature of the infection and to trace its source.

Naval Medical Compassionate Fund.

A meeting of the subscribers of this fund will be held at 3 P.M. on March 31st at the Medical Department of the Navy, Archway Block N., Admiralty, London, S.W. 1, to elect six directors of the fund.

Eugenics Society.

At a meeting of this society, to be held at 8.30 P.M. on Tuesday, March 15th, at the rooms of the Linnean Society, Burlington House, Piccadilly, W., Mr. J. Myles Bickerton will speak on Eugenics and Blindness.

Royal Statistical Society.

The *Frances Wood Memorial Prize*, value £30, is offered this year for the best investigation, on statistical lines, of any problem affecting the economic or social conditions of the wage-earning classes. Particulars may be had from the hon. secretaries of the Royal Statistical Society, 9, Adelphi-terrace, London, W.C. 2.

History of Pædiatrics.

Dr. G. F. Still will give a lecture on the Medical Care of the Child through the Ages, at 3 P.M. on Tuesday, March 15th, at 31, Belgrave-square, London, S.W. The lecture will be in aid of King's College Hospital, and particulars will be found in our advertisement columns.

Course on Disorders of Digestion.

A course on the medical and hydrological treatment of diseases of the digestive tract, the liver, and of nutrition, will be held from April 18th to May 2nd at the Hôpital Necker, Paris, under the direction of Prof. Maurice Villaret. It will include a study tour to Vichy from April 30th onwards. Particulars may be had from the Laboratoire d'Hydrologie of the Faculté de Médecine de Paris (École pratique).

A Reduced Maternity Death-rate.

At Glasgow Royal Maternity and Women's Hospital last year the maternity death-rate was 2 per cent., as against 2·6 per cent. in the previous year. The number of abnormal cases was 62 per cent. of all admissions; in the previous year the percentage was 70.

A Summer School of Individual Psychology.

The Individual Psychology Association will hold a summer school this year at Semmering, a beautiful pass in the mountains about two hours' railway journey from Vienna. The meeting will take place from June 20th to July 10th, and can be attended by English people at a reasonable cost, as the Association can obtain concessions in respect of quarters and fares for the congressists. Prospectuses and further information can be obtained from the Secretary, Mrs. Sofie Lazarsfeld, Seilergasse 16, Vienna, I.

London Fever Hospital.

At the annual meeting of this hospital recently Lord Ebury, the president, stated that there was a deficit of £2337 on the year's working, and that failure to obtain a good response to a renewed appeal "will inevitably mean closing this institution, which for 130 years has done such wonderful work, and is still necessary to the life of this great city."

Southport Hydropathic Hospital.

In 1931 the patients admitted numbered 433, of whom nearly 300 came under the general heading of rheumatism. Most of them were able to return home to resume their normal avocations. The next largest group of patients, numbering 120, were suffering from affections of the nervous system. In the great majority of sciatic cases "relief has been secured promptly, and in many has been retained permanently."

British Health Resorts Association.

This newly formed association will hold a regional conference at Hastings during the coming week-end. After a banquet on Friday night, the proceedings will open on Saturday morning with a meeting at which Dr. W. G. Willoughby, president of the British Medical Association, will take the chair, and the speakers will include Dr. E. P. Poulton, Dr. R. A. Young, Dr. Leonard Findlay, Dr. Wilfred Harris, Dr. G. R. Bruce, Dr. Percy Lewis, Dr. J. R. Dingley, and Dr. Thomas Reed. At a second meeting later in the day Lord Meston will preside, and brief addresses will be given, among others, by Lieut.-Colonel R. H. Elliot, Mr. Clough Williams-Ellis, and Dr. Fortescue Fox. Proposals will be made for the formation of a local branch of the association. On Sunday visits will be paid to Eastbourne, Bexhill, and other resorts in the area. The address of the hon. secretary of the association is 6, Elsworthy-road, London, N.W.3.

Medical Diary

Information to be included in this column should reach us in proper form on Tuesday, and cannot appear if it reaches us later than the first post on Wednesday morning.

SOCIETIES

ROYAL SOCIETY OF MEDICINE, 1, Wimpole-street, W.

TUESDAY, March 8th.—5 P.M., THERAPEUTICS AND PHARMACOLOGY. Paper: Dr. G. H. Oriel: Some Observations on the Treatment of Allergic Disease. A discussion will follow which will be opened by Prof. van Lecuwen. 8.30 P.M., PSYCHIATRY. Paper: Dr. J. Ernest Nicole: Some Sources of Misunderstanding in Psychopathology.

WEDNESDAY.—5 P.M., SURGERY: SUB-SECTION OF PROCTOLOGY. Discussion: Ano-rectal Fistula. Opener: Mr. Ernest Miles. Followed by Mr. J. P. Lockhart-Mummery and others.

THURSDAY.—8.30 P.M., UROLOGY: OBSTETRICS AND GYNÆCOLOGY. Discussion: Frequency of Micturition in Women. Openers: Mr. G. E. Neligan and Mr. Victor Bonney.

FRIDAY.—5.30 P.M., CLINICAL. (Cases at 4.30 P.M.) Cases: Mr. Cecil P. G. Wakeley: 1. Temporal Meningocele. Dr. Parsons Smith: 2. Mönckeberg's Sclerosis. Dr. Bernard Myers: 3 and 4. Thrombocytopenic Purpura. Dr. Terence East: 5. Familial Acholuric Jaundice. Dr. J. L. Livingstone: 6. Leukæmia. Mr. A. J. Watson: 7. Elephantiasis. Dr. F. Parkes Weber: 8. Erythræmia. Dr. Dorothy Hare: 9. Episcleritis and Arthritis Following Diathermy Treatment of Tonsils. Other cases will be shown. 5 P.M., OPHTHALMOLOGY. Clinical Meeting at the London Hospital, E. Cases will be shown.

MEDICAL SOCIETY OF LONDON, 11, Chandos-street, Cavendish-square, W.

MONDAY, March 7th.—9 P.M., Mr. F. A. Williamson-Noble: The Reactions of the Eye to General Disease. (Second Lettsomian lecture.)

HARLEY MEDICAL SOCIETY, 149, Harley-street, W.

THURSDAY, March 10th.—9 P.M., Inaugural Meeting. Mr. P. Lockhart-Mummery: The Origin of Tumours. To be followed by a discussion.

SOUTH-WEST LONDON MEDICAL SOCIETY.

WEDNESDAY, March 9th.—9 P.M. (at Bolingbroke Hospital, Wandsworth Common), Dr. W. J. O'Donovan: Pruritus

WEST KENT MEDICO-CHIRURGICAL SOCIETY.

FRIDAY, March 11th.—8.45 P.M. (at the Miller General Hospital, Greenwich, S.E.), Mr. T. M. Thomas: Some Surgical Emergencies of Childhood.

MEDICAL SOCIETY OF INDIVIDUAL PSYCHOLOGY, Chandos-street, Cavendish-square, W.

THURSDAY, March 10th.—8.30 P.M., Dr. Bevan-Brown: A Case of Dissociation—for Discussion.

PADDINGTON MEDICAL SOCIETY.

TUESDAY, March 8th.— 9 P.M. (at the Great Western Royal Hotel, Paddington, W.), Dr. Alfred Cox: Forty Years' Experience of Medical Organisation and its Results.

BIOCHEMICAL SOCIETY.

FRIDAY, March 11th.—3 P.M. (at University College, Gower-street, W.C.). Communications: 1. K. Culhane and G. N. Greenwood: The Activity of Crystalline Insulin. 2. J. D. Bernal (introduced by G. F. Marrian): A Crystallographic Examination of Œstrin. 3. N. K. Adam, J. F. Danielli, G. A. D. Haslewood, and G. F. Marrian: Monomolecular Films of Œstrin Derivatives. 4. E. Boyland: Creatine and Phosphorus Compounds in Tumours. 5. G. M. Richardson: The Accurate Titration of "Free HCl" of Gastric Contents, Peptic Digests, &c. 6. K. Culhane: The Importance of the Basal Diet for Vitamin A Tests. 7. K. Culhane: The Influence of Light White Casein in the Onset of Xerophthalmia.

ROYAL INSTITUTION, 21, Albemarle-street, W.

THURSDAY, March 10th.—5.15 P.M., Prof. J. B. S. Haldane: Heredity in Man.

LECTURES, ADDRESSES, DEMONSTRATIONS, &c.

ROYAL COLLEGE OF PHYSICIANS, Pall Mall East, S.W.

TUESDAY, March 8th.—5 P.M., Dr. L. J. Witts: Pathology and Treatment of Anæmia. (Last Goulstonian lecture.)

THURSDAY.—5 P.M., Dr. C. E. Lakin: The Borderlands of Medicine. (First Lumleian lecture.)

FELLOWSHIP OF MEDICINE AND POST-GRADUATE MEDICAL ASSOCIATION, 1, Wimpole-street, W.

MONDAY, March 7th, to SATURDAY, March 12th.—MEDICAL SOCIETY OF LONDON, 11, Chandos-street, Cavendish-square. Wed., Treatment series, Mr. Somerville Hastings on Mastoid Disease, at 4 P.M. (Open to Members only.)—M.R.C.P. EVENING COURSE. Mon. and Fri., at 8 P.M. at the London Temperance Hospital, Out-patient Department, Hampstead-road. Five demonstrators each evening, special cases. Wed. at 8.30 P.M., at the Medical Society lecture room, lecture by Dr. H. Gainsborough on The Significance and Measurement of Renal Efficiency. Thurs. at 8.30 P.M., at 10, Bedford-square, W.C., lecture by Dr. A. Knyvett Gordon on the Clinical Pathology of Septicæmia, illustrated by Epidiascope, followed by a Laboratory Demonstration. Open to Members only.—WEST END HOSPITAL FOR NERVOUS DISEASES, 73, Welbeck-street, W. Lecture demonstrations daily at 5 P.M.—BETHLEM ROYAL HOSPITAL, Monks Orchard, Eden Park, Beckenham. Tues. and Fri. at 11 A.M., lecture demonstration in psychological medicine.—ROYAL WATERLOO HOSPITAL, Waterloo-road, S.E. Course in Medicine, Surgery, and Gynæcology, all day.—ROYAL NATIONAL ORTHOPÆDIC HOSPITAL, Great Portland-street, W. Special post-graduate course in Orthopædics. All day. (Above courses open only to Members; apply Fellowship of Medicine.)—METROPOLITAN HOSPITAL, Kingsland-road, Demonstration of Cases in Ward by Dr. J. W. Linnell. Tues. at 2.30 P.M.

UNIVERSITY COLLEGE HOSPITAL MEDICAL SCHOOL, University-street, W.C.

TUESDAY, March 8th.—5.15 P.M., Prof. C. C. Okell: Anaphylaxis.

FRIDAY.—5 P.M., Prof. C. R. Harington: Nature of the Secretion of the Thyroid Gland (Sydney Ringer Lecture).

LONDON SCHOOL OF DERMATOLOGY, St. John's Hospital, 49, Leicester-square, W.C.

TUESDAY, March 8th.—5 P.M., Dr. H. T. Barron: Common Eruptions in Childhood.

THURSDAY.—5 P.M., Dr. J. E. M. Wigley: Pemphigus and Allied Eruptions.

NATIONAL HOSPITAL, Queen-square, W.C.

MONDAY, March 7th.—2 P.M., Dr. Riddoch: Out-patient Clinic. 3.30 P.M., Dr. Hinds Howell: Trauma in Relation to Nervous Disorders.

TUESDAY.—2 P.M., Dr. Walshe: Out-patient Clinic. 3.30 P.M., Dr. Adie: Neurosyphilis.

WEDNESDAY.—2 P.M., Dr. Martin: Out-patient Clinic.

THURSDAY.—2 P.M., Dr. Kinnier Wilson: Out-patient Clinic. 3.30 P.M., Mr. Just: Demonstration of Methods of Testing the Eighth Nerve. (In Electrical Department.)

FRIDAY.—2 P.M., Dr. Adie: Out-patient Clinic. 3.30 P.M., Dr. James Collier: Remarks upon Interstitial Neuritis.

CHARING CROSS HOSPITAL MEDICAL SCHOOL.

SUNDAY, March 6th.—10.45 A.M., Dr. H. G. E. Williams: Indications and Technique of Version in Obstetrics 11.45 P.M., Dr. J. E. M. Wigley: Infantile Eczema.

GUY'S HOSPITAL, London Bridge, S.E.

MONDAY, March 7th.—5 P.M., Dr. A. F. Hurst: Use of X Rays in Physiological Investigations.

ROYAL NORTHERN HOSPITAL, Holloway-road, N.

WEDNESDAY, March 9th.—3.15 P.M. (at the Royal Chest Hospital), Dr. N. Schuster: Demonstration of Pathological Specimens.

KING'S COLLEGE HOSPITAL MEDICAL SCHOOL, Denmark-hill, S.E.

THURSDAY, March 10th.—9 P.M., Dr. Graham Hodgson: Portable Radiology.

SOUTH-WEST LONDON POST-GRADUATE ASSOCIATION, St. James' Hospital, Ouseley-road, Balham.

WEDNESDAY, March 9th.—4 P.M., Mr. Stanley Dodd: Sterility.

ST. MARK'S HOSPITAL, City-road, E.C.

THURSDAY, March 10th.—4.30 P.M., Mr. J. K. Hasler: Low Spinal Anæsthesia.

ST. PETER'S HOSPITAL, Henrietta-street, W.C.

WEDNESDAY, March 9th.—3 P.M., Dr. C. Dukes: Tumours of the Upper Urinary Tract.

CENTRAL LONDON THROAT, NOSE, AND EAR HOSPITAL, Gray's Inn-road, W.C.

FRIDAY, March 11th.—4 P.M., Mr. W. A. Mill: Laryngitis.

HAMPSTEAD GENERAL AND NORTH-WEST LONDON HOSPITAL.

WEDNESDAY, March 9th.—4 P.M., Mr. A. Sorsby: The Ophthalmoscope in Cardiovascular Disease.

SALFORD ROYAL HOSPITAL.

THURSDAY, Marcy 10th.—Dr. H. T. Ashby: Demonstration of Diseases of Children.

NATIONAL COUNCIL FOR MENTAL HYGIENE.

WEDNESDAY, March 9th.—5.30 P.M. (at 11, Chandos-street, W.), Miss S. M. Fry: A Magistrate's Problems for Psychologists.

MANCHESTER ROYAL INFIRMARY.

TUESDAY, March 8th.—4.15 P.M., Mr. E. W. Telford: Dystrophies of Growing Bone.

FRIDAY.—4.15 P.M., Dr. T. H. Oliver: Demonstration of Medical Cases.

ANCOATS HOSPITAL, Manchester.

THURSDAY, March 10th.—4.15 P.M., Mr. H. Platt: Some Common Affections of the Hip-joint.

ST. MARY'S HOSPITALS, Manchester.

FRIDAY, March 11th.—4.15 P.M. (at the Whitworth-street West Hospital): Dr. Bramwell: The Treatment of Heart Disease in Pregnancy.

WESTERN INFIRMARY, Glasgow.

WEDNESDAY, March 9th.—4.15 P.M., Dr. J. G. Macgregor-Robertson: Venereal Diseases (Male).

NOTES, COMMENTS, AND ABSTRACTS

YOUTH AND THE CINEMA.

THE cinema makes its impression essentially upon the eye, and the majority of people have a more retentive memory for what they see than for what they hear. Again, while the cinema affects people of all ages and all classes, it makes a particularly strong appeal to children and adolescents. Sir Charles Grant Robertson, speaking from the chair at the National Conference on Problems connected with the Cinema, held at the University of Birmingham on Feb. 27th, said that within his lifetime four great developments of science—motor-cars, films, aviation, and the wireless—had fundamentally altered the lives of ordinary men and women. In his opinion the most revolutionary of the four was the cinema, and yet it was the only one left entirely without Government control in this country. Sir Charles said he made a practice of attending the cinema in the company of young people, and he could not escape a sense of misgiving at the thought of a steady infiltration into their subconsciousness of the kind of representation and criticism of life shown on the films. Lady Barrett, M.D., emphasised this point when she quoted the answer of many children at the time of the Birmingham inquiry as to what they learned from the pictures—"We learn what life is really like." Everybody would agree, she said, that it was not a good thing for children, who had no background with which to compare what they saw, to be introduced to an unworthy view of life and to form the opinion that normal life was like that. Lady Barrett dealt with the question of parental responsibility, and made an analogy with the powers of a food controller. A mother might wish to give her children food which was cheap and which did not appear to her harmful, but a food controller would confiscate it because he understood the danger of ptomaine poisoning. The average mother could not be expected to be an expert in psychology any more than in toxicology, and we had as much right to protect our children from poisoned minds as from poisoned bodies. Miss Cottell (Bristol), ex-president of the National Union of Women Teachers, reminded the Conference that we have refused to allow parents unlimited responsibility in other matters, such as whether their children shall attend school, or whether they shall engage in street trading, and there was reason to believe that children required some protection in the matter of attendance at films. As a teacher, she found that children's delicacy of taste was vitiated by over-sensationalism and over-sentimentality of the cinema. Mr. J. Lovat Fraser, M.P., in his work among young delinquents, had found undoubted evidence that the cinema encouraged a tendency to youthful offences. Dr. W. A. Potts pleaded for improvement of the cinema in the interest of adolescent boys and girls. He attended the pictures regularly once a week to see for himself the effect of the films on the minds of young people. As a medical practitioner, as a psychologist, and as a father, he felt bound to protest against scenes which appeared to be staged with a deliberate intention to stimulate the sex instinct. Life at the threshold of manhood and womanhood was difficult enough without adding to the burden of temptation.

Throughout the day a large amount of evidence was forthcoming as to a widespread dissatisfaction with the conduct of the cinema, and a real desire for its amendment. Only one resolution was on the agenda, and after much discussion it was passed in the following form: "That this Conference submits that a comprehensive inquiry into the arrangements by which films are licensed for public exhibition is urgently called for, and asks the Home Secretary to receive a deputation on this subject."

THE SCIENCE OF MEDICINE.

THE author of this book, whose full title is "The Science of Medicine: How Chronic Diseases are Cured Medicinally by Raphael Roche,"[1] informs us that he has received great commendation in the lay press, that he has made communications himself to that press on the science of medicine, and that the book is submitted as the fruit of over half a century of practice. He deprecates estimates of his medicinal cures, if formed on the fact that he employs (in his own words) "drugs in such an extreme dilution that no analysis, chemical or spectroscopic, will discover their existence"; but from instructed therapeutists he must expect such criticism. He refers to "the emphasis I lay on unusual symptoms, which may sound like affectation"; the instructed physician or surgeon knows that the unusual symptom is often the revealing one and could never find fault with the intention to detect it though he might wonder whence the diagnostic skill is derived. Attention is drawn on the cover to the fact that the work contains a full statement by Mr. Bernard Shaw of his medicinal cure by the author; the instructed doctor will here find some odd logic. The author and Mr. Reginald Dingle, who is thanked for his help in writing the book, have produced an indictment which will give pleasure to all who are "agin the doctor," and which may attract the public to the consulting room of Mr. Roche, whose fees we learn from himself, "are so much higher than those of any legally qualified physician." Of *how* diseases are cured there is nothing intelligible said; but there are allusions to Vital Force.

PSYCHOLOGY IN INDUSTRY.

IT is gratifying in these days to learn of one industrial body that has experienced no decline in its activities. The eleventh report of the National Institute of Industrial Psychology describes 32 investigations in factories, shops, and offices. Whereas in the past attention has chiefly been centred on problems of production, it seems probable that the future may see the same intense thought and activity applied to problems of distribution; the selection and training of salesmen, the display of goods, market research, and sales organisation. As examples of the Institute's work may be cited a demonstration of the superiority of hand-packing over conveyor packing in a chocolate factory, whereby a saving of £2000 a year was effected; and the reorganisation planned for a patent flour factory which will save £3700 a year on an initial outlay of £7000. Further investigations have been carried out for the Post Office; tests have been devised for phonogram and teleprinter candidates; and various forms, previously misunderstood by many recipients, have been redrafted to save correspondence and explanation. During 1931 the Institute enrolled 284 new members and received more than 7000 visitors, including the Prince of Wales. Public demonstrations are given on the last Wednesday in each month, and on at least one Saturday morning, when films are shown and methods demonstrated. The Journal has become so popular that it is to appear monthly instead of quarterly, under the title of *The Human Factor*, and early in 1932 a popular survey ("Industrial Psychology in Practice") and a history of the Institute ("Ten Years of Industrial Psychology") are to be published.

The vocational guidance department advised 500 young people during the year under review, and the cases examined in 1927 and 1928 have been followed up by a questionaire. Of 90 people working in accordance or apparently in accordance with the Institute's advice, 76 were found to be successful

[1] By Raphael Roche. London: C. W. Daniel Company. 1932. Pp. 159. 5*s*. net.

and only eight definitely unsuccessful. Of 44 who did not seem to have taken the advice, 17 were successful and 17 unsuccessful, with ten doubtful. This department is also working among Borstal lads. A great deal of research is being done in the vocational department as well as along more general lines. The work of secretaries and nurses is being analysed; the nature and measurement of the mental abilities involved in factory assembly operations have been investigated; experiments in typewriting have shown the great importance of rhythm in developing speed; and occupational conditions among the blind have been studied. More and more the Institute is making itself an essential and integral part of the industrial life of the nation.

INCUNABULA IN THE R.C.S. LIBRARY.

Mr. W. R. le Fanu, librarian to the Royal College of Surgeons of England, has compiled a list of the Incunabula in the College library [1] containing over fifty valuable items, but the experienced reader's eye will linger over three or four jewels. There is Benevenuto Grassi's De Oculis, 1474, recently translated by Casey A. Wood; John of Gaddesden's Rosa Anglica; Petrus de Abano's Conciliator differentiarum philosophorum et medicorum, &c., 1476; and Michael Scotus's Liber Physionomiæ, 1490. Osler wrote of Michael Scot as one of the greatest thirteenth century scholars, with whose observations on the temperaments and on the significance of dreams in persons of different complexions Freud and the modern advocates of incubation sleep would sympathise. It might be hoped that some day the breath of life which animates the museum below might stir the library to demonstrate its treasures to a wider circle.

TREATMENT OF RHEUMATOID ARTHRITIS.

WE are informed by the Anglo-French Drug Co., Ltd. (238A, Gray's Inn-road, London, W.C.), that they hold stocks of Allochrysine, the gold compound mentioned by Dr. Jacques Forestier in his paper on rheumatoid arthritis in our last issue.

Vacancies

For further information refer to the advertisement columns.

Aberdeen City District Asylum, Newmachar.—Jun. Asst. P. £300.
Abergavenny, Monmouthshire Mental Hospital.—Jun. Asst. M.O. £350.
Ascot, Heatherwood Hospital for Orthopædic Children.—Res. Med. Supt. £850.
Aylesbury, Royal Buckinghamshire Hospital.—Res. M.O. £200.
Barnstaple, North Devon Infirmary.—Res. M.O. At rate of £150.
Beit Memorial Fellowships for Medical Research.—Jun. Fellows. Each £400.
Bradford, Royal Eye and Ear Hospital.—Jun. H.S. £150.
Bradford Royal Infirmary.—H.S. At rate of £135.
Bristol Eye Hospital.—Hon. Asst. Surgeon. Also Asst. Res. H.S. £100.
Canterbury, Kent and Canterbury Hospital.—H.P. At rate of £125.
Cardiff City.—Asst. M.O. £500.
Cardiff City Mental Hospital, Whitchurch.—Clin. Asst. and Research Student. £200.
Cardiff, Welsh National School of Medicine.—Jun. Asst. in Medical Unit. £250.
Chester, East Lanchashire Tuberculosis Colony, Barrowmore Hall, Gt. Barrow.—H.P. At rate of £100.
Coventry and Warwickshire Hospital.—Res. H.P. and H.S. £160 and £125 respectively.
Derby, Derbyshire Hospital for Women, Friar Gate.—H.S. At rate of £150.
Doncaster Royal Infirmary.—Sen. Cas. H.S. At rate of £175.
Dover, Royal Victoria Hospital.—H.S. £150.
Durham County Hospital.—Two H.S.'s. Each £150.
East Ham Memorial Hospital, Shrewsbury-road, E.—H.P. and H.S. and Cas. O. At rate of £150 and £120 respectively.
Golden-square Throat, Nose, and Ear Hospital, W.—Hon. Anæsthetist.
Great Barrow, Nr. Chester, East Lancs., Tuberculosis Colony.—Asst. M.O. At rate of £275.
Greenwich, Dreadnought Hospital.—H.P. and H.S. Each at rate of £110.
Grocers' Company, Grocers' Hall, E.C.—Research Scholarships. Each £300.
Halifax, St. Luke's Hospital.—Sen. Res. M.O. £350.
Hampstead General and N.W. London Hospital, Haverstock Hill, N.W.—Cas. Surg. O. for Out-patient Dept. At rate of £100.
Harrogate Infirmary.—Sen. H.S. At rate of £150.
Hereford, Herefordshire General Hospital.—Hon. Anæsthetist.
Hertford County Hospital.—H.S. At rate of £200.
Hospital for Consumption and Diseases of the Chest, Brompton, S.W.—Whole-time Research Worker. £350. Also Med. Reg. £200.
Hospital for Sick Children, Great Ormond-street, W.C.—Cas. O. £400.
Hospital for Tropical Diseases, Endsleigh-gardens, W.C.—H.P. At rate of £150.
Ilford, King George Hospital.—Cas. H.S. At rate of £100.
Infants Hospital, Vincent-square, Westminster.—H.P. At rate of £75.
Ipswich, East Suffolk and Ipswich Hospital.—H.S. and H.P. Each £120.
Isleworth, West Middlesex County Hospital.—Res. Asst. M.O. £400.
Lancaster County Mental Hospital.—Asst. M.O. £500.
Leeds, St. James's Hospital.—H.P. At rate of £150.
Liverpool, County Mental Hospital, Rainhill.—Asst. M.O. £500.
Liverpool, David Lewis Northern Hospital.—Hon. Asst. Surg.
London Homœopathic Hospital, Great Ormond-street, W.C.—Phys.
London Hospital, E.—Hon. Clin. Asst.
Macclesfield General Infirmary.—Sen. and second H.S.'s. At rate of £180 and £150 respectively.
Manchester Babies' Hospital, Burrage-lane, Levenshulme.—Jun. Res. M.O. At rate of £50.
Manchester, Royal Children's Hospital, Pendlebury.—Res. M.O. At rate of £125.
Manchester, Royal Children's Hospital, Gartside-street.—Two Asst. M.O.'s. Each at rate of £150.
Manchester Royal Infirmary.—Med. Reg. to Out-patients. At rate of £150.
Manchester and Salford Hospital for Skin Diseases.—Asst. M.O. £100.
Margate, Royal Sea Bathing Hospital.—Asst. Med. Supt. £500.
Miller General Hospital, Greenwich-road, S.E.—Anæsthetist. 52 gns. Also H.P. £100.
Northampton General Hospital.—H.P. and three H.S.'s. Each at rate of £150.
Norwich, Norfolk and Norwich Hospital.—H.P. £120.
Nottingham and Midland Eye Infirmary.—Res. H.S. £200.
Parbold, Lancs, Wrightington Hospital.—Jun. Asst. M.O. £200.
Portsmouth Royal Hospital.—H.S. At rate of £130.
Princess Louise Kensington Hospital for Children, St. Quentin-avenue, N. Kensington, W.—H.P. At rate of £75.
Queen's Hospital for Children, Hackney-road, E.—H.P. and Cas. O. Each at rate of £100. Also Surgeon to Ear, Nose, and Throat Dept.
Royal Dental Hospital of London, 32, Leicester-square, W.C.—Asst. Dental Surgeon. Also House Anæsthetist for In- and Out-patient Department. 10s. 6d. per session.
Royal National Orthopædic Hospital, 234, Gt. Portland-street, W. H.S. At rate of £150. Also 2 H.S.'s for Country Branch, Brockley Hill, Stanmore. Each at rate of £150.
Royal Northern Hospital, Holloway, N.—Res. M.O. £200. Also H.S. At rate of £70.
St. Alfege's Hospital, 48, Vanbrugh Hill, Greenwich, S.E.—Res. Asst. M.O. £350.
St. Bartholomew's Hospital.—Dental H.S. £80.
St. Helens County Borough.—Deputy M.O.H. £700.
St. Leonards-on-Sea, Buchanan Hospital.—Jun. H.S. At rate of £125.
St. Peter's Hospital for Stone, &c., Henrietta-street, Covent Garden, W.C.—H.S. At rate of £75.
Salford Royal Hospital.—Res. H.S.'s to Special Depts. Each at rate of £125.
Scarborough Hospital and Dispensary.—Two H.S.'s. Each at rate of £175.
Southampton, Royal South Hants and Southampton Hospital.—Hon. Asst. Surg.
Stafford, Staffordshire General Infirmary.—H.P. At rate o £150.
Stoke-on-Trent, North Staffordshire Royal Infirmary.—Orthopædic H.S. £150.
Surrey County Mental Hospital Service.—Jun. Asst. M.O. £350.
Swanley, Kent, Hospital Convalescent Home, Parkwood.—Res. M.O. At rate of £200.
Taunton, Taunton and Somerset Hospital.—Sen. House M.O. and H.S. £150 and £100 respectively.
Wakefield Clayton Hospital.—Two Jun. H.S.'s. Each at rate of £150.
Walsall, General Hospital.—H.P. and Res. Pathologist and H.S. Each at rate of £150.
Warwick, King Edward VII. Memorial Sanatorium, Hertford Hill.—Jun. M.O. At rate of £200.
West London Hospital, Hammersmith-road, W.—H.P. and H.S.'s., H.S. to Ear, Nose, and Throat Dept., and Res. Anæsthetist. Each at rate of £100. Also Hon. Med. Radiologist.
Wigan, Royal Albert Edward Infirmary and Dispensary.—Three H.S.'s. Each at rate of £150.
Willesden General Hospital, N.W.—Cas. O. At rate of £120. Also H.S. and H.P. At rate of £110 and £100 respectively.
York Dispensary.—Res. M.O. £175.

The Chief Inspector of Factories announces vacant appointments for Certifying Factory Surgeons at Holsworthy (Devon), Barrowden (Rutland), Boroughbridge (Yorks, W.R.), Creech St. Michael (Somerset), Cranbrook (Kent), and Troon (Ayr).

The Secretary of State for the Home Department gives notice of vacancies for medical referees under the Workmen's Compensation Act for the Bishops Castle, Bridgnorth, Llanfyllin, Ludlow, Madeley, Oswestry, Shrewsbury, Wellington, Welshpool, and Whitchurch County Court Districts. Applications should reach the Private Secretary, Home Office, Whitehall, London, S.W., not later than March 23rd.

[1] Reprint from Annals of Medical History, N.S. vol. ii., pp. 674–76.